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OBSERVATIONS

ON THE

TREATMENT OF CHLOROSIS BY HYDROCHLORIC ACID.

By W. HALE WHITE, M.D.

Few facts in therapeutics are more certain than that iron cures chlorosis; and yet, as is the case with so many other of our really valuable remedies, we are unable to explain how it acts. Inasmuch as the most constant symptom of chlorosis is a deficiency of iron in the blood, it was for a long time assumed that the iron given as a medicine was absorbed into the blood, and that there making good the deficiency, it cured the disease. But a little thought will show serious objections to such a view.

In the first place, the blood in chlorosis is deficient, not only in iron, but also in red blood-corpuscles. What corpuscles there are have too little iron in them; it is possible that adding iron to the blood might increase their iron until it reached for each corpuscle the healthy amount, but we do not know why iron should lead to the development of new corpuscles. Again, in any case of chlorosis the amount of iron the body lacks is only a few grains. Suppose the healthy weight of a woman who becomes the subject of chlorosis to be nine stone, that is 126 pounds, she will have in health about nine and a half pounds of blood. The greater part of this is water; not all the solids are hæmoglobin. Hæmoglobin is a very complex molecule, for it contains more than 2000 atoms ($C_{712}H_{1130}N_{214}O_{245}FeS_2$), and of these only one is iron. Therefore it follows that the amount of iron in the blood is very slight. Schmidt has calculated that it is about 0·05 per cent. of the weight of the whole blood, which would allow a woman of nine stone thirty-three grains of iron in her blood. Now, supposing that she is moderately ill with chlorosis, she will have lost half the iron from the blood, or about sixteen grains of iron; so that if she took five grains of reduced iron three times a day, she would have taken enough iron in a single day to raise the amount in the blood to the healthy standard. But we all know from experience that chlorosis cannot be cured in a day; large doses of iron have to be taken every day for a month at least in many cases.

Then, also, if it were a mere question of putting in iron, which, being absorbed, directly improved the blood, there ought to be no necessity for iron to be given as a medicine, for ordinary diet contains quite enough. Dr. Stevenson has been good enough to supply me with the following analysis of the Guy's Hospital full diet. A day's allowance contains 0·45 grains of iron in the 12 oz. of bread, 0·15 grains in the half-pint of milk, 0·05 grains in 4 oz. of mutton, 0·10 grains in half a pound of rice pudding, and 0·15 grains in 8 oz. of potatoes; the butter contains none, or the merest traces; therefore this daily diet contains 0·9 grains of iron. Consequently, a chlorotic girl who is sixteen grains of iron short would, in two and a half weeks, take enough iron to make good the deficit in the blood; but we all know that it will not cure chlorotic patients to put them on full diet without any medicine at all. I have tried this in two cases, which will be found in the appendix to this paper, and it will be seen that they hardly improved at all on full diet only. They will be alluded to in detail presently.

The other reasons why it is impossible to look upon the cure of chlorosis as simply a question of the addition of iron to the blood are—first, that it is often necessary, in order to prevent a relapse, to continue the iron even after the red colour has returned to the cheeks; secondly, the fact that the cure in some cases can be effected, or at any rate accelerated, by arsenic; thirdly, it has been shown by MM. Pecholier and Saint-Pierre[1] that salts of copper will cure chlorosis; and fourthly, that chlorosis can probably be cured by permanganate of potassium. It might be thought that possibly the arsenic, the copper, or the manganese combined with some substance in the body which contained iron, and that in so doing it set free the iron; but it is very difficult to believe that arsenic could turn out iron from any compound. Thus, for clinical reasons, we are forced to conclude that iron does not cure in chlorosis by merely directly making up the amount of iron of which the blood is short.

It has always been taught, and is still stated in many books, that when iron is given internally an albuminate is formed, and it is in this state absorbed. Let us see if this is true. Hamburger[2] has made experiments to decide whether iron is absorbed at all from the intestine. A dog, which weighed eight kilogrammes, was given 300 grammes of meat in a day containing 15 milligrammes of iron. It had thus taken 180 milligrammes of iron in twelve days. Of this amount 38·4 appeared in the urine, 136·3 in the fæces, 1·8 in the bile; altogether 176·5 milligrammes. During the subsequent nine days 49 milligrammes of iron a day, in the form of sulphate of iron, were added to the previous diet. At the end of these nine days the first allowance of meat was resumed for four days more. Thus, in thirteen days, 195 milligrammes of iron were taken in the meat, and 441 as sulphate of iron; altogether 636 milligrammes. The quantity excreted amounted to 58·4 in the urine, 549·2 in the fæces, 0·8 in the bile; altogether 608·4. Therefore, during twelve days on which no sulphate of iron was being given, the amount of iron excreted in the urine was 38·4 milligrammes, and during nine days during which, on the same diet, sulphate of iron was given, and during the four days subsequent to this, or thirteen days in all, the amount excreted in the urine was 58·4 milligrammes. So that the administration of sulphate of iron by the mouth leads to very little more iron in the urine, but an enormous increase of it in the fæces. A second experiment gave similar results. These experiments appear to show that very little of the sulphate of iron taken is absorbed, the greater part passing out in the fæces as the sulphide and turning them black.

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[1] 'Revue Générale de Clinique et de Thérapeutique,' Jan. 7th, 1891.

[2] Hamburger, 'Zeitschr. f. physiol. Chem.,' vol. ii, p. 191, 1878; and Bunge, 'Physiol. and Path. Chem.,' trans. by L. C. Wooldridge, p. 95.

There are, however, two possibilities: it may be that the iron is absorbed, and again re-excreted into the intestine; or it may be that it is absorbed and locked up in some organ in the body.

Now, to determine if iron is excreted into the intestine from the blood, the first thing is to decide whether in a fasting animal, or in one being fed on iron-free food, any iron is excreted into the intestine. Bidder and Schmidt, forty years ago, found in the urine of a fasting cat from 1·4 to 1·7 milligrammes of iron daily, and from six to ten times the amount in the fæces. Gottlieb[1] cleared out the intestine of a dog, weighing 9 kilogrammes, with sulphate of magnesium. He then fed it on a diet of 100 grms. of starch paste, 50 grms. of suet, and 50 grms. of curds a day, and he emptied the bowels daily with sulphate of magnesium; the fæces contained 3·5 milligrammes of iron a day. He calls this a diet very poor in iron, but he admits that it contains 1·5 to 2·0 milligrammes of iron in a day. These experiments, he considers, show that iron can reach the intestines by excretion from the blood, but that the quantity is very small. This might be poured in either through the bile or the gastric and intestinal juice. But these experiments are not worth so much as they appear at first sight to be, for Gottlieb himself admits the diet contains iron, and this is confirmed by the following analysis, with which Dr. Stevenson has kindly supplied me. One pint of milk contains ·048 grain of iron, and it yields 13½ fluid ounces of whey, which contain ·023 grain of iron; thus, when milk is curdled, half the iron goes into curds and half into the whey. Thus some of the small amount of iron in the fæces might have come from the food; and as regards the rest, it must be remembered that the intestinal epithelial cells which are constantly being shed contain much iron.

Many experimenters have stated that the bile contains iron, and most text-books have copied this statement, which, however, is probably incorrect, for Bunge, Hamburger, and other of the more recent investigators have been unable to detect more than the merest traces of iron in the bile. Speaking of the iron found in the intestine of fasting animals and those fed on a diet nearly free from iron, Bunge says, "It may be that iron is excreted through the intestinal wall, perhaps by the aid of leucocytes, which appear to play an active part in excretion as well as in absorption. To me the most natural explanation seems to be that the iron found in the excreta of fasting animals arises from the rejected epithelial cells of the intestine. The dried epithelium of the intestine contains, according to C. Schmidt's analysis, 0·46 per cent. more iron than the hæmoglobin itself."

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[1] 'Zeitsch. f. physiol. Chemie,' Band xv, Heft 5.

These experiments are, then, unsatisfactory, for they do not teach us whether the intestine can excrete iron from the blood, but the balance of evidence is that it does not.

The attempt has been made to determine the question by injecting large amounts of iron into the circulation or under the skin, and observing the quantity in the intestine. All observers are agreed that injection of iron in this way leads to a very trifling excess in the amount excreted in the urine, so if the iron is not excreted into the intestine, it must be locked up in the tissues of the body. Among the older experimenters, Buchheim and Mayer, after injecting salts of iron into the blood, found a large amount in the intestine; but Quincke, after the injection of lactate of iron into the jugular vein, did not obtain any excess in the intestine. Jacobi and Gottlieb are the two most recent experimenters on the subject, but they are likewise at variance. Jacobi fed dogs on milk, and every day cleared out the bowels with Carlsbad salts; by this means he reduced the amount of iron in the fæces to almost nothing. He then injected into the femoral vein a solution of a tartrate of iron which contained in 1 c.c. 0·0065 grm. of iron; the amount of fluid injected was equivalent to 0·143 grm. of iron. The dog was soon after bled to death. The abdomen was at once opened, and the intestines were sprayed upon with an ether spray to contract the vessels and drive out the blood from them. He then found that the wall of the large intestine contained 1·9 milligrammes of iron, that of the small 4·9; the contents of the large 1·0 milligramme, and of the small 1·28. The total was 9·08 milligrammes, or only 6·35 per cent. of what he injected. In a second experiment, conducted in the same way, he only recovered 15·1 per cent. of the amount injected. Neither he nor Novi had been able to find that the injection of iron into the vessels causes any appreciable increase in the bile. Jacobi, therefore, concludes that after the intra-venous injection of iron the amount in the intestines, bile, and urine is so slightly raised that the iron must be stored up somewhere. Gottlieb's[1] results are directly contrary to these. He cleared out the intestine with Glauber's salts, kept the dogs starved for many days, and then fed them on the diet which, as has already been mentioned, he considers sufficiently free from iron. He analysed the fæces daily for six days, and found that 3·5 milligrammes of iron was the average passed every day. He then injected iron subcutaneously for nine days, the total amount injected being 100 milligrammes. During these nine days the daily average excretion of iron was 6·8 milligrammes of iron in the fæces. During the subsequent nineteen days the average daily amount of iron in the fæces was 6·6 milligrammes; it then fell to normal. He reckons that without any injection of iron the amount in the fæces would be in twenty-eight days 86·2 milligrammes, but that for the twenty-eight days, during nine of which 100 milligrammes of iron was injected subcutaneously, and the nineteen subsequent days, the total amount of iron in the fæces was 183·1 milligrammes, so that the subcutaneous injection of 100 milligrammes of iron increased the amount of the iron in the fæces by 96·2 milligrammes. Other experiments gave the same results, namely, that after the injection of large quantities of iron into the veins, a very considerable proportion, often as much as three quarters of it, could be recovered from the intestine. Further, at the time of death the liver contained so much iron that it is extremely probable that the iron, in its way through the body, is temporarily stored up in the liver, and from there passed on into the intestine.

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[1] 'Zeitschrift f. physiol. Chem.,' Bd. xv, Heft 5.

These experiments appear to render it probable that iron can be excreted from the blood into the intestine, and they are supported by the oft-quoted experiments of Cahn, who injected salts of manganese into the blood of rabbits, and found that manganese reappeared in the contents of the stomach and intestine. I do not think, however, that too much stress should be laid upon the experiments of Gottlieb, for Jacobi obtained contrary results, the value of which is, however, diminished, as he killed his animals too soon; still, even allowing that some iron in Gottlieb's experiments was excreted into the intestine, it is a long step to conclude, from the fact that when salts of iron are suddenly thrown into the blood they reappear in the intestine, that salts of iron given by the mouth are probably absorbed into the blood and re-excreted into the intestine. We have just seen that it is highly improbable that inorganic salts of iron are absorbed from the intestine; secondly, if they are, it by no means follows that they are absorbed as inorganic compounds, at all comparable in their action to the salts of iron that Gottlieb and others have thrown into the veins. Further, all the recent work appears to show that animals differ very much in their behaviour to iron. Bunge, in discussing this question, draws attention to the fact that sometimes, after injection of salts of iron into the blood, symptoms of poisoning, such as reduced blood-pressure, intestinal disturbances, and paralysis of the central nervous system, appear, but that these symptoms are never observed after the introduction of iron into the stomach. He suggests that this shows that the iron is not absorbed; and it certainly seems to me to prove that large amounts of inorganic compounds of iron cannot in man be absorbed into the blood, for I have sometimes given such large doses of perchloride of iron in cases of gastric hæmorrhage that the patient has taken a gramme of iron in a few hours, but none of these symptoms of poisoning have occurred. But even, for the sake of argument, allowing that inorganic iron compounds can be absorbed, it by no means follows that they are re-excreted into the intestine, for although manganese salts injected into the blood are excreted into the intestine, yet, if they are taken into the stomach, no evidence of their absorption can be discovered.

Still it is just possible that the iron may be absorbed in an innocuous form; or that after absorption, while in the body, it may be converted into some innocuous compounds of iron quite incapable of producing poisoning symptoms.

The outcome of all we have said is this, that the commonly received doctrine that the inorganic salts of iron are absorbed is not proved, nor is it disproved, but on the whole the balance of evidence renders it very probable that they are not absorbed. Nor is it settled whether, if they are absorbed, they may not be re-excreted into the intestine. Some evidence here points one way, some the other. If we assume that the inorganic iron compounds are not absorbed from the intestine, how then is it possible that iron can cure chlorosis?

Whatever salt of iron is introduced into the stomach, it quickly becomes converted into a chloride. The ferric chloride, when it comes in contact with the sodium carbonate in the intestinal juice, becomes an oxide of iron, and the ferrous chloride becomes ferrous carbonate. Both the oxide and the carbonate are soluble in the presence of the organic substances present in the intestine. Later on, by the action of the sulphur compounds, and the reducing agents in the intestines, these iron compounds are converted into the sulphide of iron, and as such excreted with the fæces.

Bunge has proposed the following hypothesis to meet these facts. He shows that all the iron present in our food exists in very complex organic compounds, of which hæmatin is a well-known example. On the supposition that inorganic salts cannot be absorbed, we must conclude that iron is only capable of absorption in the form of these complex organic compounds. He shows that sulphide of ammonium can slowly remove the iron from these complex organic compounds, and convert into an inorganic sulphide. Now, he supposes that in chlorosis too little hydrochloric acid is secreted by the stomach, and as a result indigestion is present, as indeed it commonly is. The hydrochloric acid is powerfully antiseptic, consequently without it fermentative processes are very rife, and micro-organisms flourish. The micro-organisms he supposes, especially those of butyric acid fermentation, are so abundant that they pass into the small intestine. As a result of the butyric acid fermentation, hydrogen is set free, and alkaline sulphides are formed by the reducing action of this nascent hydrogen on the sulphur compounds of the food. These alkaline sulphides, acting like the sulphide of ammonium, decompose the complex organic iron compounds in the food, and therefore there are none of them to be absorbed. It is supposed that the reason why iron cures chlorosis is that the iron salts administered as medicine are acted upon by the alkaline sulphides, and that consequently the iron in the food is protected. This doctrine is, he considers, supported by the fact that iron is only beneficial in large doses, for a large quantity is required for the alkaline sulphides to act upon in order that they may not act on the iron in the food.

Now I come to the work that I have done with the object of testing the truth of this hypothesis. The first thing to see was what improvement patients would make who were only put upon hospital full diet, which, as we have already seen, contains quite enough iron to cure them. All the patients were put to bed and kept there for the sake of uniformity of conditions. The following table shows the result of treating two girls with chlorosis with rest in bed and full diet. They are Cases 1 and 2 in the appendix.

	Increase on 2 to 3 weeks' full diet, no iron as medicine.	Increase on 13 days' treatment with sulphate of iron and full diet.
Case 1.—Red corpuscles	18 per cent.	4 per cent.
Hæmoglobin	20 ,,	34 ,,
Case 2.—Red corpuscles	32 ,,	14 (a little before it was 26)
Hæmoglobin	3 ,,	65 per cent.

Thus we see that, even on full diet without any iron as a medicine, the number of red corpuscles increased considerably, and that there was some increase of hæmoglobin. In the first case this increase was just about as much as was proportional to the increase in the number of corpuscles, so here it is clear that the richness of each corpuscle remained about the same as before the investigation began. In the second case the number of the corpuscles increased largely; in fact, they reached almost the number of health, but the hæmoglobin hardly increased at all, and therefore the new corpuscles must have had very little hæmoglobin in them. We may, therefore, conclude that rest in bed and full diet will increase the number of red blood-corpuscles considerably, but the individual richness does not increase, and it may even fall.

When the patient was put upon sulphate of iron the quantity of hæmoglobin at once increased very largely, but the increase in the number of red corpuscles was comparatively slight. It therefore appears that rest in bed and good diet lead to the formation of red corpuscles, but that the administration of iron rapidly leads to the formation of fresh hæmoglobin. We may, I think, conclude that there is no evidence in these observations to show that in chlorosis the iron in the full diet is absorbed when no iron as a drug is administered.

The theory that hydrochloric acid in the stomach is deficient in chlorosis would receive much support if it could be shown that this disease could be cured by the administration of hydrochloric acid. I have treated five cases in this way.

They are Observations 3, 4, 5, 6, and 7 in the appendix to this paper, and they are tabulated on p. 11.

All the patients were kept in bed and on full diet during the whole of the observations. They said they felt better for the treatment by hydrochloric acid, but their colour did not improve, and it is quite possible that the diet and rest in bed were responsible for their feeling better. With regard to the blood, it will be seen that upon the acid treatment, which was always tried for a long time, in some of the cases as long as a month, the red corpuscles increased 20, 22, 4, 6, and 38 per cent., but the hæmoglobin only increased 15, 8, 5, 5, 5 per cent. Thus it is clear that under hydrochloric acid there is often a very considerable increase in the number of corpuscles, but the increase in the amount of hæmoglobin is trifling, so that individual richness of the corpuscles in hæmoglobin is much less than it was before the acid was given. These patients, in fact, did no better than those who were treated by rest and full diet only.

Upon the iron treatment, in two cases in which the corpuscles had upon the acid treatment reached nearly 100 per cent., their number fell a little; and in the other three the increase was 10, 23, 32 per cent., but the increase in the hæmoglobin was as follows (in Obs. 7 there is no record of the hæmoglobin after the iron, but, as the patient's colour was much improved, we may fairly set the increase down as the same as in the previous three cases): 45, 32, 40, 35, 35; that is to say, there was always a great and rapid increase in the amount of hæmoglobin.

	On admission.	After treatment by hydrochloric acid.	After treatment by iron salts.	Drugs given. Acid means hydrochloric acid.
Case 3—				
Red corpuscles, per cent.	79	80 (99)	100	Acid for a month.
Hæmoglobin „	35	50	95	Iron sulphate for a month. Under the acid the corpuscles reached 99, but fell again to 80.
Case 4—				
Red corpuscles, per cent.	64	86	96	Acid for a month.
Hæmoglobin „	35	43	75	Carbonate of iron 5 days.
Case 5—				
Red corpuscles, per cent.	59·4	63	86	Acid taken for 14 days, pushed to 75 ♏ a day. Perchloride of iron 8 days.
Hæmoglobin „	30	35	75	
Case 6—				
Red corpuscles, per cent.	52	58	90	Acid taken 3 weeks.
Hæmoglobin „	35	40	75	Large doses of perchloride of iron for a month.
Case 7—				
Red corpuscles, per cent.	56	94	86	Acid taken 3 weeks.
Hæmoglobin „	35	40	Not taken, but colour very much improved	Large doses of sulphate of iron for 3 weeks.

We may conclude, therefore, that hydrochloric acid has no more efficacy in curing chlorosis than has simple rest in bed and good food. Whether the hydrochloric acid is given, or whether it is omitted, there is usually a considerable increase in the number of red corpuscles, but very little improvement in the amount of hæmoglobin. The patient feels better under both circumstances. But directly large doses of iron are given there is a rapid increase in the amount of hæmoglobin, and the patient begins to get well quickly. These experiments, therefore, negative the supposition that the cause of chlorosis is a deficiency in the secretion of hydrochloric acid by the gastric juice. I should have liked to perform the crucial experiment of feeding a patient suffering from chlorosis upon a diet free from iron, and giving her some inorganic salt of iron at the same time; but this is impossible, for one cannot construct a diet containing no iron. The conclusions which I would draw from this paper are—

(1) Hydrochloric acid will not cure chlorosis. Patients treated with it do not improve more than they would do with rest in bed and good food.

(2) Rest in bed and good food cause in chlorosis a considerable increase in the number of red corpuscles, but only a slight increase in the amount of hæmoglobin.

(3) The sulphate, or the chloride, or the carbonate of iron leads to a rapid cure of chlorosis.

(4) A diet containing curds is not an iron-free diet.

APPENDIX OF CASES.

Two Cases of Chlorosis treated without Drugs.

Obs. 1. *Chlorosis treated without drugs.*—Amy McA—, æt. 14, admitted 6th May, 1891, into Mary Ward. (Reported by Mr. O. C. Worthington.)

Previous and family history.—Unimportant.

Eight months ago began to have pain in chest, fainting, indigestion, shortness of breath.

On admission.—Pale, constipated. Basic hæmic murmur, bruit de diable. Hæmoglobin 20 per cent. Red corpuscles 40 per cent.

May 26.—Has been treated with Aq. Menth. Pip., purgatives as required, and full diet. Hæmoglobin 40 per cent.

Red corpuscles 58 per cent. To take a pill of Ferri Sulph. Exsic. gr. ij, Ext. Nucis Vom. gr. ¼, Conf. Ros. q. s., thrice a day.

30th.—Two pills thrice a day.

June 7th.—Is much better. Colour good, but hæmic murmurs remain. Hæmoglobin 74 per cent. Corpuscles 62 per cent.

Left the hospital.

Obs. 2. *Chlorosis treated without drugs.*—Ellen D—, æt. 16, admitted into Mary Ward May 6th, 1891. (Reported by Mr. F. P. S. Cresswell.) Always had good health till six months ago, when she began to lose colour, to become giddy, and to suffer from fainting fits, dyspnœa, constipation, nausea, amenorrhœa, and dyspepsia.

On admission.—She is very pale and white. There is a basic hæmic murmur, and a venous hum over the jugular veins. Headache and constipation are present. Red corpuscles 60 per cent. Hæmoglobin 22 per cent. Ordered Pil. Col. c̄ Hyoscy. gr. x st., and M. M. c̄ M. S. p. r. n., Aq. Menth. Pip. ℥j, t. d. s. Full diet.

May 13th.—Feels much better, no headache; basic murmur not so loud, and venous hum gone.

21st.—Red corpuscles 92 per cent. Hæmoglobin 25 per cent.

22nd.—Ferri Sulph. Exsic. gr. ij, Ext. Nucis Vom. gr. ¼, Conf. Ros. q. s., t. d. s.

June 1st.—Red corpuscles 118 per cent. Hæmoglobin 73 per cent.

7th.—Red corpuscles 106 per cent. Hæmoglobin 90 per cent. Has got her colour back, and feels quite well. Left the hospital to-day.

FIVE CASES OF CHLOROSIS TREATED WITH HYDROCHLORIC ACID.

Obs. 3. *A case of chlorosis treated by hydrochloric acid.*—Sarah P—, æt. 18, admitted into Mary Ward October 1st, 1890. She came in for fainting fits and pains in the head. She had been in Miriam Ward in April, 1890, for anæmia and occasional blueness and coldness of the fingers, being almost a mild form of Raynaud's disease. The red corpuscles were then 60 per cent. of normal, and the hæmoglobin 30 per cent. She recovered on treatment by iron, and remained well till the middle of August.

On admission.—Skin and visible mucous membranes all very pale. Bowels regular, no vomiting. Respiration hurried. Cardiac dulness, and position of apex-beat normal. There is a loud hæmic murmur, loudest in the pulmonary area. Urine normal. No optic neuritis. Menstruation regular. No œdema of legs. Complains of buzzing in the ears and occipital headache. Hæmoglobin 35 per cent. Red corpuscles 79 per cent. She was ordered dilute hydrochloric acid ♏x, syrup ʒj, water to ℥j, t. d. s.

October 10th.—Patient better; 45 per cent. of hæmoglobin.

21st.—Red corpuscles 99 per cent. Hæmoglobin 45 per cent. Patient is much better, and her colour has improved; the buzzing and headache have disappeared, but the systolic murmur remains, and she is troubled with constipation.

November 3rd.—Red corpuscles 80 per cent. Hæmoglobin 50 per cent. She is better in every way; there is no hæmic murmur, and she does not suffer from constipation. As the hæmoglobin is still very deficient, she was ordered Ferri Sulph. Exsiccat. gr. ij, Ext. Nucis Vomic. gr. $\frac{1}{4}$. One pill ter die for three days, two pills ter die for the next three days, three pills ter die afterwards.

14th.—She seems very well. Red corpuscles 99 per cent. Hæmoglobin 65 per cent.

December 2nd.—Patient seems perfectly well; she was discharged cured. Red corpuscles 100 per cent. Hæmoglobin 95 per cent.

Obs. 4. *Chlorosis treated by hydrochloric acid.* (Reported by Mr. A. H. Godson.)—Ellen W—, æt. 17, a general servant, was admitted into Mary Ward January 26th, 1891. The family and personal history are unimportant. The patient suffers from shortness of breath, weakness, headache, vomiting, constipation, and amenorrhœa. She is very pale and sallow, and has a hæmic murmur. Blood: Red corpuscles 64 per cent.; hæmoglobin 35 per cent.

January 30th.—Acid. Hydrochlor. dil. ♏x, Syrup. ʒj, Aq. ad ℥j, t. d. s.

February 4th.—Red corpuscles 72 per cent. Hæmoglobin 35 per cent.

17th.—Red corpuscles 78 per cent. Hæmoglobin 40 per cent.

27th.—She says she feels very much better. Red corpuscles 86 per cent. Hæmoglobin 43 per cent. To take Pulv. Ferri Carb. Sacch. gr. xv ter die.

March 4.—Feels much better. Red corpuscles 96 per cent. Hæmoglobin 75 per cent.

11th.—She went out to-day, very much improved.

Obs. 5. *Chlorosis treated by hydrochloric acid.* (Reported by Mr. W. G. Roper.)—Harriet S—, æt. 30, admitted 4th March, 1891, into Miriam Ward.

Family history unimportant. The patient has always suffered from amenorrhœa. *Present illness.*—The amenorrhœa has been increasing the last few years. Lately she has begun to feel weak, to lose colour, to suffer from palpitation, nausea, sickness, giddiness, singing in the ears, swelling of the ankles, and constipation. *On admission.*—She is of a pale greenish-yellow colour, there is a loud hæmic murmur. The blood is thin and watery. It gives the following result upon examination: Red corpuscles 59·4 per cent.; hæmoglobin 30 per cent. Ordered Acid. Hydrochlor. dil. ♏x, in syrup and water, thrice daily; middle diet.

March 12.—She says she feels better, but her colour has not improved. An examination of the blood gave red corpuscles 78 per cent.; hæmoglobin 35 per cent. To take Acid. Hydrochloric. dil. ♏xx, thrice a day.

14th.—To take Acid. Hydrochloric. dil. ♏xxv, thrice a day. She feels much better, but does not look less anæmic.

17th.—Red corpuscles 63 per cent. Hæmoglobin 35 per cent.

18th.—The patient says she feels very much better, and her appetite has improved, but as neither her colour nor the condition of her blood had altered, the hydrochloric acid was discontinued and the following substituted: Tinct.

Ferri Perchlor. ♏xx, Mag. Sulph. ʒss, Syrup. Limonis ʒij, Aq. ad ℥ss, 6tis horis.

26th.—She has in every way much improved. She has a much better colour, and the blood which flows when her finger is pricked is natural-looking. Red corpuscles 86 per cent. Hæmoglobin 75 per cent.

28th.—To take her medicine every four hours.

31st.—Left the hospital to-day.

Obs. 6. *Chlorosis treated by hydrochloric acid.* (Reported by Mr. C. F. Wakefield.)—Emily C—, æt. 26, admitted into Mary Ward January 14th, 1891. Is a general servant in a public-house. Family history and previous history unimportant. Has been getting a pale yellow colour for the past year, and the other symptoms which have gradually supervened have been palpitation, dyspnœa, swelling of the feet, constipation, pain after food, vomiting, and buzzing in the ears. There is a hæmic murmur and a venous hum in the neck. The blood showed red corpuscles 52 per cent. of normal. Hæmoglobin 35 per cent.

January 17th.—Middle diet, and began to take the following: Acid. Hydrochlor. dil. ♏x, Syrup. ʒj, Aq. ad ℥j, t. d. s.

30th.—Feels better in herself, but does not appear so.

February 6th.—Red corpuscles 58 per cent. Hæmoglobin 40 per cent. Ordered Ferri Sulph. Exsiccat. gr. j, Ext. Nucis Vomicæ gr. $\frac{1}{4}$, as a pill; one thrice a day for the first three days, two thrice a day for the second three days, three thrice a day for the third three days.

24th.—Is much better in every respect; no hæmic murmurs. Red corpuscles 84 per cent. Hæmoglobin 65 per cent. To take four pills thrice a day.

March 6th.—Left the hospital. Red corpuscles 90 per cent. Hæmoglobin 75 per cent. Is much better. Commenced to menstruate two days ago.

Obs. 7. *Chlorosis treated by hydrochloric acid.* (Reported by Mr. T. S. Robson).—Julia C—, æt. 16, admitted into Mary Ward January 14th, 1891. She is a general servant; her previous and family histories are unimportant.

Her symptoms, which began eight months ago, are principally the following:—Shortness of breath, swelling of feet, constipation, nausea, and amenorrhœa.

On admission.—She was very pale and anæmic, and had a hæmic murmur. Blood: Red corpuscles 56 per cent.; hæmoglobin 35 per cent. To take Acid. Hydrochlor. dil. ♏x, Syrup. ʒj, Aq. ad ℥j, ter die.

January 24th.—Red corpuscles 86 per cent. Hæmoglobin 40 per cent.

February 4th.—Red corpuscles 94 per cent. Hæmoglobin 40 per cent. To take Ferri Sulph. Exsiccat. gr. j, Ext. Nucis Vom. gr. ¼. One three times a day for three days, two three times a day for next three days, and three three times a day afterwards.

12th.—Red corpuscles 86 per cent. Hæmoglobin 45 per cent.

26th.—Is very much better in every way. Red corpuscles 86 per cent. Her improvement in colour is very marked.

March 1st.—Left the hospital.

ON SOME

INTERESTING PATHOLOGICAL CONDITIONS MET WITH IN ANIMALS,

FROM THE POST-MORTEM ROOM OF THE ZOOLOGICAL GARDENS.

By H. J. CAMPBELL, M.D.

By the kindness of the Zoological Society, and of Mr. Beddard, I have for some time past had the opportunity of making post-mortem examinations of animals dying in the Zoological Gardens. I propose in this paper giving a short account of a few cases which appear to me to be interesting. Some of the conditions described are very rare in man. On the other hand, others strikingly resemble those found in young children.

Tuberculosis is by far the commonest cause of death. Out of one hundred and twenty-six consecutive cases I have found it present, in one form or another, in forty-one, occurring with the greatest frequency in monkeys and birds, and amongst the latter most frequently in vegetable feeders.

Out of the thirty-eight monkeys on which I made post-mortem examinations, twenty showed tuberculous lesions; and in most of these the tuberculosis was general, the lungs, liver, and spleen being the organs most commonly affected. One striking fact I observed in the tuberculous affections of monkeys was the great similarity in the pathological changes met with in them and in young children. This similarity is not confined to very young monkeys, but, as far as my limited experience goes, is found in monkeys of all ages.

Thus, in the lungs, a caseous broncho-pneumonia is exceedingly common, rendering the whole or the greater part of one or more lobes quite solid; whereas cavitation, though occasionally present, is rare. Again, in several cases the disease had apparently spread from a bronchial gland directly to the adjacent lung. Again, as in children, the roots of the lungs were not infrequently found to be the parts most affected.

The following are my notes of the conditions present in a few cases.

In a Malbruck monkey the right pleura was much thickened, and the right lung had hardly any healthy tissue left, it being one mass of patches of softening caseous material. The disease had apparently originated in caseating glands at the root of the lung; these glands, which consisted of soft breaking-down caseating material, had encroached on the lung, and had directly affected it. At its apex the left lung was quite free from disease, but at its base there were several patches of caseating broncho-pneumonia. At the bifurcation of the trachea a caseous gland had suppurated, and had burst into the œsophagus.

In a Rhesus monkey I found extensive disease in both upper lobes; the left lower lobe consisted of a solid caseous mass; the base of the right lower lobe was studded with small patches of breaking-down caseous material; the visceral surfaces of both pleuræ were covered with miliary tubercles; the bronchial and cervical glands, and also those along the aorta, were caseating.

In another Rhesus monkey there was, at the upper and posterior portion of the left lower lobe, a soft breaking-down mass, separated from the lung by a fibrous capsule, which was evidently a suppurating gland.

In a Macaque monkey the left lower lobe was solid throughout, being in a condition of caseous pneumonia. In the right lung, and in the left upper lobe, there were several patches of caseous broncho-pneumonia. The apices were free from disease.

In another case I found a large cavity at the apex of the left lung, while the left lower lobe was solid.

The following case of a Rhesus monkey is peculiar, the conditions met with differing in many respects from those usually found in monkeys. There was recent pleurisy over the whole surface of the left lung, while at its root there were a number of breaking-down patches of caseous broncho-pneumonia. At the apex of the left lower lobe there was a ragged-walled cavity, as big as a walnut, and filled with pus. The extreme apices of both lungs were free from disease, but there was caseation in the lobes on the left side; it was more extensive in the upper than in the lower lobe. The right lung was studded all over with grey miliary tubercles, but there was no tubercle to be found in any other part of the body. The peculiarities of this case, therefore, consisted in the greater implication of the left upper lobe than of the lower, in the greater tendency to cavitation than is usual, and in the absence of tubercle in the liver and spleen.

In a Malbruck monkey I found double tubercular pleurisy, with a localised empyema at the dorsal and lower portion of the left chest. The lower third of the upper lobe of the left lung was entirely transformed into a caseous mass; there were no cavities, but caseous patches were dotted here and there in both lungs. The apices were free; the bronchial glands were swollen and caseous.

In another case I found left-sided pleurisy. The lung was firmly adherent to the chest-wall anteriorly and inferiorly; posteriorly it was free above, but adherent below. The upper half of the upper left lobe was converted into a ragged-walled cavity, which was filled with pus; the lower half of the upper lobe was solid, being in a condition of caseous broncho-pneumonia. The lower lobe on the left side, and the whole of the right lung, were studded with grey miliary tubercles, and dotted with numerous small foci of suppuration. These small abscesses were surrounded by lung tissue in a condition of grey hepatisation.

Thus we see that, as regards the lungs, there is a great similarity between the conditions frequently observed in children and in those present in the above-mentioned cases. But there is a marked dissimilarity in the comparative infrequency of any disease being met with in the brains of monkeys, either in the form of a tubercular meningitis or in that of a caseous nodule. The heart and pericardium are very rarely affected. In one case which came under my notice, that of a pig-tailed monkey, there were two small tubercular nodules on the parietal surface of the pericardium.

On the other hand, as in children, the spleen is affected in a very large number of cases; I found it to be so in thirteen out of the twenty monkeys I examined. The organ was generally much enlarged, with an irregularly nodulated surface. It was occasionally affected with miliary tubercle, but was more frequently studded with caseous nodules. In some cases there was hardly any healthy tissue left, almost the whole organ being changed into a caseous mass. In other cases there were several cavities, containing purulent fluid, so that the condition shown, on cutting across the organ, could be fairly described as cavitated. Thus in a Rhesus monkey the spleen was much enlarged, and nodular on the surface, and contained several large cavities, together with a number of caseating nodules. In another very similar case the spleen was also much enlarged, and contained numerous cavities; the greater part of the organ was changed into a caseous mass, so that only about one tenth of it presented normal characteristics.

The liver was found to be affected even more often than the spleen—in fact, in fourteen out of the twenty cases that came under my notice. In one it was studded throughout with grey miliary tubercles, in another it contained a large number of small caseous nodules, and in a third it was riddled with small caseating tubercles.

In only three cases did I find the peritoneum to be affected.

In one of these, the chimpanzee, "Sally," I found very extensive tubercular peritonitis; the intestines were all matted together, and the mesentery much contracted; numerous caseating glands were present, and there was a small quantity of fluid in the abdominal cavity. There was also fluid in both chests, and recent lymph on the surface of both lungs. I found also a few miliary tubercles and several patches of broncho-pneumonia in the lungs, but the affection of these organs was evidently of very recent date. The liver and spleen both contained a few tubercles, and there were tubercular ulcers in the intestine. In only a very few other cases did the intestine show typical tubercular ulceration.

In one Rhesus monkey the uterus showed two ulcers on its inner surface, while the cavity contained about an ounce of breaking-down blood-clot. There were several tubercles in the substance of the muscle.

The lymph-glands very frequently showed caseous changes, but the kidneys rarely; in only a few cases did they present a few grey miliary tubercles. In no case did the supra-renal capsules show any disease.

From these cases, and from others which I have not mentioned, one may, I think, draw the following conclusions: that cavitation is rare; that caseous broncho-pneumonia is the commonest condition; that the apices, instead of being especially liable to the disease, are generally free or less affected than other parts; that the lungs are rarely alone affected; that in most cases the liver and spleen are attacked; that in a few the peritoneum is affected; and that in a large number caseating glands are present.

Tuberculosis, though most prevalent in monkeys, is also very frequently found in many other animals, especially in birds. Thus I have found it in a globose curassow, in a lineated kallego, in two pheasants, in three ducks, in a white-tailed eagle, in a touracou, in a pigeon, in a lark, in a kingfisher, in a parrot, in an eagle owl, in a cormorant, in a finch, in a mynah, and in a rhea.

In the curassow there was a mass of caseating material about the left knee-joint, the synovial membrane was pulpy and much thickened, and the cartilages were eroded. Old calcareous nodules were present in the bases of both lungs, while the liver and spleen contained numerous miliary tubercles.

In the white-tailed eagle the spleen was enlarged and nodular. It might be described as consisting of a mass of tubercular caseating material. There were also scattered tubercles in the liver.

In the lineated kallege the liver was studded all over with patches of breaking-down caseating material, and the peritoneum was dotted with miliary tubercles. In the place where the left testis ought to have been, there was a large mass of tubercular caseous material in which not a trace of testicular structure remained; no left testis could be found, although the right was in its normal position and of normal size.

In the ring-tailed pheasant the liver and spleen were studded with caseous nodules, the greater part of the normal structure of both organs being replaced by caseous material; the peritoneum contained many tubercles.

In the summer duck both lungs showed many patches of caseous broncho-pneumonia, and the liver was studded with recent miliary tubercles.

In the pigeon, the liver, spleen, and left lung showed acute miliary tuberculosis, while the left anterior and posterior thoracic air-sacs were affected with an acute tubercular inflammation.

In the short-toed lark I found a fatty-looking tumour round one elbow-joint. It was soft but not fluctuating, and only thinly covered over by skin. On cutting into it I found that it consisted of solid caseating material; tubercle bacilli were present in great abundance. The interior of the joint and all the internal organs appeared to be perfectly healthy.

In the Indian tree duck the lungs were healthy, except for one caseating nodule as big as a pea, which I found in the right lung. Cheesy lymph was present on the peritoneal membrane, and a little serous turbid fluid in the abdominal cavity. The liver was quite solid, and retained its shape as though it had been moulded in plaster. Its tissue was largely replaced by a firm ? caseous material. There were a few caseous nodules on the peritoneal surface of the intestine, but there was no ulceration of the mucous membrane corresponding to the nodules on the external surface.

In the vinaceous Amazon parrot there was at the base of the tongue a large tubercular caseating mass, which was as large as a small almond, and which projected into the pharynx. There was also another caseating mass, in front of the vertebral column, between the œsophagus and the bone, while in the pectoralis major there was an encapsuled pea-sized nodule.

In the eagle owl, at the bifurcation of the trachea I found a caseating mass, which had involved and perforated the wall, and had nearly blocked up both bronchi. The lungs were somewhat collapsed, but in no part airless. There were a few caseous deposits in the liver about the portal fissure and on the surface of the spleen; the other organs were all healthy.

In the cormorant the right lung was largely affected. About two thirds of its normal tissue was replaced by a mass of caseating cheesy material, mixed with altered blood. The air-sac in connection with this part of the lung was filled with a yellow cheesy mass, while its walls were thickened. The liver and spleen were riddled with small tubercular nodules.

In the finch both lungs were so filled with caseous patches, that there appeared to be hardly any healthy tissue left. The liver also contained much caseous material, which replaced the normal tissue.

In the mynah the liver showed many caseous nodules, while all the other organs were healthy.

In the rhea the intestines were in a very remarkable condition. Along their whole length, and in both cæca, there were a very large number of caseous-looking tumours, which projected into the lumen of the gut; many of these were ulcerated on the surface, and of a black colour. The size of these tumours varied from that of a pea to that of a walnut. In no case was there any indication of their breaking down; on the contrary, they were very firm and dense. In one cæcum an intussusception, through which only a small probe could be passed, had been produced by one of these nodules having been grasped and passed on. Miliary tubercles were present in the liver, but the rest of the body was free from tubercle.

In only four other cases have I found tubercular disease, namely, in a Burrhal wild sheep, in a binturong, in an agouti, and in an Esquimaux dog.

In the Burrhal wild sheep I found extensive cavitation at both apices, and at the ventral borders of the lungs. Both lungs were closely adherent anteriorly to the dorsal surface of the sternum. There was no sign of pleurisy over any other part of the lungs, except that the pericardium was closely adherent to the ventral border. Early pericarditis was present, the heart being covered with soft œdematous lymph. In the posterior lobes there were numerous patches of broncho-pneumonia in the stage of red hepatisation, while in the anterior lobes there were older patches, already grey. The animal was very much wasted. The only other disease to be found was old capsulitis of the spleen. The chief points of interest in these cases were the limitation of the pleurisy to the most dependent region, and the marked affection of the apices of the lungs.

In the binturong the lungs contained a considerable number of small caseating nodules; both posterior lobes were collapsed, owing to the presence of a considerable quantity of serous fluid in both chests. Close to the head of the pancreas, and pressing on the inferior vena cava, there were a number of caseating glands, some of which were suppurating. Numerous caseating nodules were present in the liver, and there was one in the spleen. The abdomen contained a small quantity of serous fluid.

In the agouti there were two small caseous masses in one lung, and the liver and spleen were both in a condition of miliary tubercle.

In the Esquimaux dog there were a few tubercles on the visceral surface of the pleura, but there were none in the tissue of the lung. The small intestine showed numerous caseating nodules on its peritoneal surface; these were most plentiful at the commencement of the jejunum. This case is interesting from the fact that Professor Semmola, in a paper communicated to the Royal Medico-Chirurgical Academy of Naples, which was abstracted in the Supplement to the 'British Medical Journal' of August 22nd, 1891, stated that the dog is absolutely insusceptible to tuberculosis. It is without doubt rare to find tubercular disease in carnivorous animals. This is curious when we consider how frequently it occurs in herbivorous animals, whose raw flesh is so constantly eaten by Carnivora.

After tuberculosis, intestinal diseases were the most frequently met with. Very often enteritis was the only pathological condition to be found. In these cases the lower part of the small intestine and of the colon contained light yellow, frothy, liquid fæces, the mucous membrane was somewhat congested, and Peyer's patches and the solitary follicles were somewhat swollen. Sometimes early broncho-pneumonia, collapse at the bases or other parts of the lungs, or a little bronchitis was also present; in fact, just the conditions so frequently found in children.

I have also found intussusception. It occurred in a boa constrictor; the intussuscepted portion measured three inches, and had advanced to three inches from the anus. The gut was permeable, but only to a small probe; the part above the intussusception was dilated, and contained some liquid fæces and much mucus.

I found obstruction, due to fæcal impaction, in four cases. In a rhea I found in the lower part of the small intestine a mass of matted hair, wool, and vegetable fibre, which distended and completely occluded the gut; the coils above were much distended, and those below collapsed and empty.

In an armadillo, which had been given milk and pounded or minced meat to eat, and which had in addition eaten some of its bed, I found a little hay in the stomach, and a considerable mass of hay, partially digested, obstructing the duodenum, about six inches from the pylorus. Above the obstruction the duodenum was much dilated, and in a condition of acute inflammation; below it the intestine was much contracted, and almost completely empty. All the other organs were in a normal condition.

In a helicoderma there was an obstruction in the small intestine, about ten inches from the pylorus. It was caused by impacted food, amongst which were some pieces of egg-shell. The duodenum was much dilated above the obstruction, and its wall much thickened; below it was empty and contracted. I could not discover any perforation, but there was universal acute peritonitis, and multiple abscesses of small size were present in the liver.

I found in a water-viper a very peculiar condition, due to the presence of tapeworms, about midway between the stomach and the anus; the intestine for about six inches was much distended, and was crowded with a felt-like mass of tapeworms; the mucous membrane in this part, and above it up to the stomach, was in a condition of intense inflammation. It was swollen and red, with many submucous hæmorrhages.

The small intestine of a crane, which had been for forty-two years in the Gardens, was, for about eighteen inches of its length, crowded with ascarides; the gut had a rope-like feel, and the intestine was so choked up that it must have been quite impossible for anything but liquid substances to have passed through.

In a python the small intestine contained about fifty tapeworms (*Solenophorus megalocephalus*), which were mostly attached for about eight inches of its length to the mucous membrane: a few nematode worms were present in the lungs.

In a Macaque monkey the cæcum was distended with hard fæces. The mucous membrane showed patches of ulceration over its whole surface; the appendix, however, was healthy, and there was no trace of peritonitis.

Broncho-pneumonia is not at all uncommon. It is generally associated with some other condition, such as enteritis, &c. &c.

I have twice found empyema, once in a sheep and once in a monkey.

In the sheep it was secondary to a bedsore on the sternum. About the middle of the ventral surface of the bone there was a sore, covered with a mass of hair and sloughing skin. The bone was bare and superficially necrosed, presenting a worm-eaten appearance. Within the thorax, extending up to the apex, opposite to the bedsore, there was a localised empyema. The ventral borders of both lungs were adherent to the sternum by a mass of cheesy-looking material. Broncho-pneumonia was present in the right upper lobe of the lung, the tissue of which was breaking down in several places, forming a number of small caseating patches.

In the monkey there was a double tubercular pleurisy, with a localised empyema, on the anterior surface of the left lung.

Pleurisy with effusion and without any other disease is very rare. I found it, however, in two Diana monkeys. In the one there was effusion into the right chest, with recent lymph on the surface of the lung. In the other the pleurisy was right-sided, the whole lung was firmly adherent to the chest wall, except for a narrow strip along the dorsal surface parallel to the vertebral column. Both lungs were very œdematous; there was recent acute pericarditis, and the testes were enlarged and fibroid. There was no trace of tubercle to be found anywhere.

On the other hand, bronchitis is very common. In one case, that of a brown bear, there were several subpleural ecchymoses, and the fluid in the tubes was blood-stained.

In an Upland goose, around the root of each lung there was an enchondromatous-looking growth, infiltrating the lungs to a considerable extent. These growths were exceedingly hard, and nodular on the surface. A mass of the same consistency and appearance as the lung tumour, and about as big as a pea, was encapsuled in the pectoralis major muscle. When examined microscopically the tumours were seen to be composed of exceedingly minute cells.

Inflammation of the air-sacs is not uncommon in birds. I found an interesting condition in two gulls. In both, the walls of the interclavicular anterior thoracic air-sacs were much thickened. In the one case both air-sacs contained mould; in the other the anterior thoracic was free, but the interclavicular was lined with a thick mycelium of *Penicillium glaucum*, with abundant aërial hyphæ bearing conidia.

Diseases of the heart and pericardium are rare; I have only found endocarditis in two cases. In the one, that of a ring-tailed coati, there were recent vegetations on the edges of the mitral valve, and a breaking-down ante-mortem clot at the apex of the left ventricle. There was also double hydrothorax. In the other, that of an African rhinoceros, the endocarditis was secondary to pyæmia caused by a large bedsore on the hip. The disease was right-sided, the tricuspid valve being covered with vegetations. A mass of fibrinous clot was attached to the endocardium, opposite to the valve.

I found pericarditis in four cases—in a deer, in a squirrel, and in two monkeys. In one of the monkeys, as I have already mentioned, it was associated with right-sided pleurisy, and in the deer with general pyæmia.

Non-tubercular peritonitis is not uncommon. I found it in five cases, of which the four following are the most interesting.

In a lesser white-nosed monkey there was general acute peritonitis, which had apparently arisen from a very acute enteritis. I could not discover any intestinal perforation, and the peritonitis was not purulent. Along the whole length of the small intestine the Peyer's patches were swollen and superficially ulcerated. The ulcers were most abundant in the jejunum, and in the lower part of the duodenum; they were less plentiful near the ileo-cæcal valve, and looked older, being greyish in colour, not so much swollen, and nowhere presenting separating sloughs. The cæcum contained one patch of ulceration. At the ileo-cæcal valve itself the mucous membrane was swollen and inflamed, but not ulcerated. The small intestine was distended by a considerable quantity of blood-stained fluid, hæmorrhage having apparently taken place from some of the ulcers. The cæcum and large intestine contained soft fæces; the solitary follicles of the large intestine were swollen, but not ulcerated; the spleen was large and soft.

I also found peritonitis in two deer. In the one it was acute and universal, and was associated with a ventral omental hernia; and in the other there was pelvic cellulitis, associated with prostatic abscess.

The last case was that of a bird. Its gizzard was perforated by a fish-bone, the point of which had passed through the abdominal muscles, but had not perforated the skin; the gizzard was firmly joined to the abdominal parietes by lymph; all the tissues between the fascia and the skin were infiltrated with a greenish purulent substance of a very fœtid odour. There were no abscesses in any of the internal organs.

I have had the opportunity of examining only one case of renal calculus. It occurred in a horse. The calculus, which was of pure carbonate of lime, was at the apex of the kidney, and was surrounded by a firm capsule, which had replaced the kidney tissue.

Rickets is very common, but in only one case was it at all acute. This was in a white-nosed monkey, in which all the bones were exceedingly soft and pliable; the femora and humeri could be bent beyond a right angle without breaking; both the tibiæ and fibulæ were so softened that they had bent at right angles, so that the animal had walked on a surface made up of the foot and about a third of the leg. The joints were all much enlarged. The skull was so soft that, after the brain had been taken away, the parietal eminences could be made to meet; at the same time it was so elastic that they at once resumed their normal shape when the pressure was removed. Enteritis was also present, and the lungs were much collapsed at the bases. No hæmorrhages had taken place into the subcutaneous tissues.

Various other conditions have been present as causes of death.

In the case of a red-throated diver there were numerous ulcers with sloughy bases in the trachea. One of these, at the bifurcation, had perforated, and had set up gangrenous inflammation at the roots of both lungs.

A white-tailed eagle had swallowed a large bone about five inches long, apparently part of a tibia; this had lodged in the stomach and œsophagus, which were greatly distended by it, and from which it could not be removed.

A common sheldrake apparently committed suicide on account of the death of its mate. The trachea and larynx were completely blocked by a considerable amount of mud.

Parasites were not so common as I should have expected. This is doubtless due to the care exercised in the management of the animals. They were present, however, in a few cases.

There were a few pentastomata in the peritoneal cavity of a Malbruck monkey, which died of general tuberculosis. I frequently found ascarides, and tapeworms are not uncommon in snakes. Hydatids also are common, but in only one case amongst those I examined had they anything to do with the fatal result; this was the case of a camel, in which hydatid cysts were present in large numbers in the lungs, liver, spleen, and kidneys. The size of the cysts varied from that of a walnut to that of a small orange. Some of them were filled with a cretaceous calcifying material, others with a gelatinous substance, which contained daughter cysts. Death was due to pulmonary embolism. A cyst in the liver having apparently suppurated and opened into the vena cava inferior, the right heart was found to be filled with a mixture of this cretaceous material and blood, which also filled and occluded the pulmonary artery.

In a wallaby the liver contained two thin-walled cysts and one thick-walled one, all containing flukes; the walls of the thick-walled cyst, moreover, were undergoing caseous degeneration. The same organ also contained a number of small caseous-looking nodules, in none of which could I find any trace of the parasites which had apparently caused them. Many of the mesenteric glands were soft, and breaking down. All the other organs were in a normal condition.

External parasites are of course extremely common, but the following case of a cormorant which died of general tuberculosis is interesting from the fact that the acari had penetrated into the intermuscular connective tissue. The bird, on being opened, had a musty smell, caused by the presence of an enormous number of dead acari in the fascia covering the thigh muscles, and in the intermuscular connective tissue.

ON THE LOCALISATION OF PHTHISIS TO THE APEX OF THE LUNG.

By H. J. CAMPBELL, M.D.

The question "Why does phthisis begin at the apex?" has received many answers; but the very number and variety of these answers afford sufficient proof that the cause of this phenomenon is not very evident, and that so far no completely satisfactory explanation has been given to account for the constancy with which the earliest symptoms of the disease occur in this region.

Strümpell considers the cause to be that the apices move much less on respiration than other parts of the lung, a favorable resting-place being thus afforded for tubercle bacilli.

Ziegler expresses the same view, but further adds that the amount of blood in circulation in the apex is less than that in the rest of the lung. Hence any bacilli which may reach the lung by respiration are less readily carried off by the lymphatics, and destroyed by the living tissue cells; or, in other words, the tissue is less resistent. Also, residues of previous inflammation linger longer in the apex than elsewhere, and thus cause a kind of local weakness or predisposition to bacillary invasion.

Rühle, in Ziemsson's Cyclopædia, quotes the following statements of Freund ('Der Zusammenhang gewissen Lungen-Krankheiten mit pimären Rippen Knorpelanomalien') that the limited respiratory movements of the apex are sometimes due to early ossification of the cartilages; and further, that in cases of recovery from disease of the apex it has been frequently found that the hindrance to respiration has been removed by a fracture, and by the consequent formation of a false joint, the necessary mobility of the first rib being thus restored.

According to Rindfleisch, "if we can show that the catarrhal secretions of scrofulous persons are less easily removed from the bronchi of the apices, we can offer some explanation of the fact that the tubercular process usually begins there;" and further, "the more freely the respiratory movements are made, and the more a sufficient hyperæmia is present to mix the thick cellular secretion with serum, so much the less likely will the secretion be to remain in such a part of the lung." He thinks that these conditions are present in the apices of the human lungs in consequence of the upright position of the body, owing to which the weight of the dependent arms interferes with the respiratory movements. This fact, it seems to me, concerns rather the frequency of the occurrence of phthisis in the upper lobes than its commencement in the apices. He goes on to state that the almost exclusive occurrence of phthisis in the human race is explained by this interference with the respiratory movements of the upper part of the chest; but it is a well-known fact that phthisis is by no means confined to the human race,—in fact, as I mentioned in my previous paper, monkeys in the Zoological Gardens die far more frequently from phthisis than from any other cause. He further considers it to be probable that in feeble persons the secretions at the apices of the lungs are inspissated, as, according to Virchow, "the thickness of a catarrhal secretion, especially of the bronchi, is in inverse relation to the amount of blood in the part;" and that in such persons, owing to the poverty of the blood, and to the elasticity of the walls of the vessels when the body is upright, the greater part of the blood will be in the lower lobes, and a far less proportion in the apices.

Coupland considers that phthisis commences at the apices because they are more exposed to the influence of external cold.

In examining the lungs of monkeys that have died of phthisis, I have been much struck by the fact that the apices, instead of being more liable to the disease than other parts of the lung, are as a rule less affected. This led me to compare the anatomy of the region occupied by the apices in monkeys with that of the same region in man, to see if any difference exists which would throw light upon the subject in question. I observed that in monkeys, owing to the length of the first rib, and more especially to the backward direction of the clavicle, the entrance to the thorax is so blocked that it is impossible for the apex of the lung to rise out of the thoracic cavity. The arrangement of these parts is well shown in the accompanying drawings. The first (Fig. 1) is of a dissection of this region in a macaque monkey, the second (Fig. 2) of the skeleton of a cebus monkey; the drawing of the same region in man (Fig. 3) is introduced for the purpose of comparison.

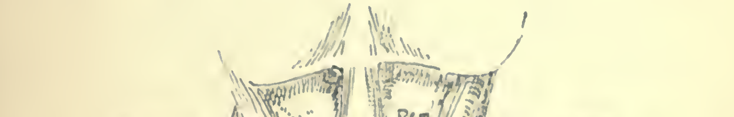

FIG. 1.

In order to demonstrate that in these monkeys the apex cannot rise out of the thorax, I forced air through the trachea into the lungs of a macaque monkey, having previously exposed the region above the clavicle. I then found that although the lungs were expanded to their fullest extent, there was absolutely no bulging of the apex into the neck above the clavicle. On repeating this experiment upon man, I found that the apex could easily be made to rise for a considerable distance above the clavicle.

In these monkeys, therefore, the whole of the lung is intra-thoracic. Also, as I have before stated, the apex is not especially liable to disease. The conclusion that I came to on considering these two facts together was, that the chief reason for the great frequency with which the apex is first attacked in man is to be found in the fact that in him the extreme apex is extra-thoracic and not intra-thoracic.

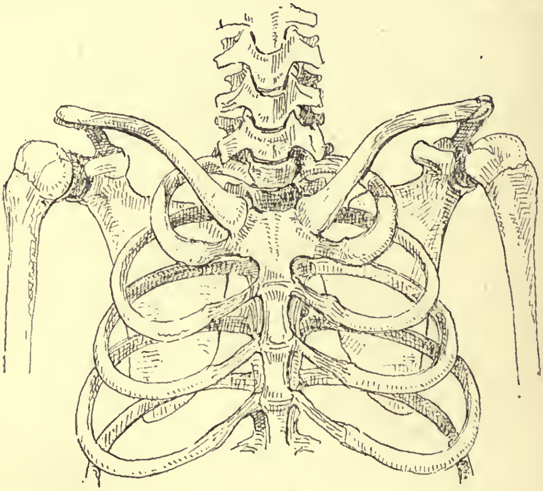

FIG. 2.

Throughout this paper I have not considered the localisation of early phthisis in children, as they need to be looked at from quite a different standpoint, owing to the frequency with which the primary focus of disease is in a bronchial gland, the readiness with which the walls of the thorax yield to pressure, and the many other points in which they differ from adults.

The extreme apex in man, being extra-thoracic, is under exactly opposite conditions, as regards pressure, to the rest of the lung; on inspiration the intra-thoracic lung is under a pressure less than that of the atmosphere, but the apex is subject to the atmospheric pressure which is communicated to it by the soft parts covering it. Thus it is forced downwards into the thoracic cavity, with the result that it becomes slightly compressed, and air is forced out of it; this air meets the incoming air from the trachea and large bronchi, and the pressure becomes equalised. The result of this is, that on inspiration only a small quantity of oxygenating air reaches the apex. Again, on expiration, the pressure on the apex being that of the atmosphere, and hence less than that on the rest of the lung, air rich in carbon dioxide, but poor in oxygen, is forced into it. From the above it follows that the contents of the apex are under most unfavorable conditions for becoming aërated, and thus the vitality of its tissues is lessened, and hence their resisting power is lowered. Further, the same conditions which interfere with the elimination of the carbon dioxide also operate to prevent secretions and excretions from finding their way out of the apices; and this retention, and the resulting decay and decom-

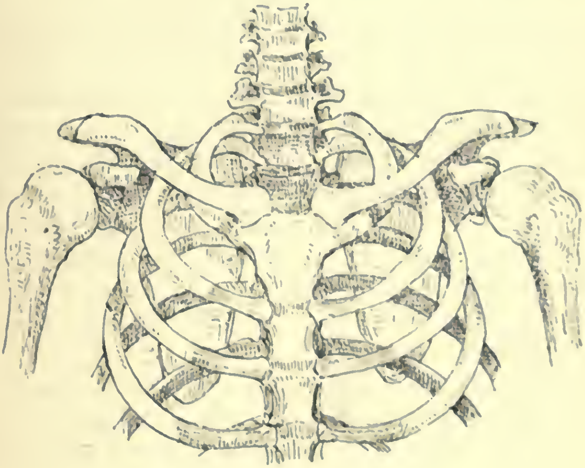

Fig. 3.

position of waste products, must still further lessen the power of resistance of the tissues to the attacks of bacilli.

It may be objected that, under the conditions above described, it would be difficult for tubercle bacilli to get into the apex. This objection, however, I think, cannot be maintained, for the apex is by no means under unfavorable conditions for getting air in, but only for receiving air rich in oxygen; in fact, during forcible expiration, as has been noticed by Rühle, air from the rest of the lung with any bacilli it may contain is driven into the apices. Again, if for any reason the glottis be closed, and the muscles of expiration be forcibly brought into play, as during violent muscular effort, coughing, &c., air is driven with such force into the extra-thoracic portions of the lung that they are seen to rise considerably in the neck. This is often seen, in an exaggerated degree, in patients with chronic bronchitis; and though less evident in healthy persons, it can generally be made out. I have attempted to graphically demonstrate the fact that the apices move in an opposite direction from the intra-thoracic lung, by means of tracings taken with Marey's tambours, but I have found it so difficult to eliminate the movements of the sterno-mastoid muscle and of the clavicle, &c., that I have been unable to obtain completely satisfactory results.

The above considerations seem to me to lend support to my view, that the localisation of early phthisis to the apices can be most satisfactorily accounted for by the fact that these parts are extra-thoracic. The upright position of the body is also an important factor, but is not sufficient alone to account for the conditions met with.

Dr. Kingston Fowler's observation that "the extreme apex of the lung is not often the site of the primary lesion, but that phthisis begins most frequently at a point from one inch to one and a half inches below the summit of the lung," seems to me to lend considerable support to my view; for although this region is not placed in quite such an unfavorable position for obtaining oxygenated air as the extreme apex, yet, as this is exactly the point of junction between the extra- and intra-thoracic portions of the lung, as has been shown by Braune by means of frozen sections, the lung at this point is always approximately in a condition of stasis, being constantly subjected to equal pressure from either side. Hence it will be just in this region that secretions, bacilli, &c., will lodge, and where they will be in the most favorable position for remaining at rest.

If this view be correct, it is evident that the contraction which takes place in the apex in phthisis is of the greatest value for the purpose of cure, for by its means the extra-thoracic portion may be drawn down so as, eventually, to become intra-thoracic. Should this occur, the improved conditions under which the apex is placed must materially increase the chances of an active phthisis becoming a healed phthisis. If it were possible to hasten this desirable result by excising the extreme apex, it is probable that, in cases of early phthisis, cure would take place far more frequently than it does at present. The cases so frequently seen in the post-mortem room, where the scarring and contraction of the apex show that phthisis has been present but has disappeared, are probably cases in which this retraction of the apex has taken place.

Again, the view that the onset of emphysema in a patient with early phthisis is not infrequently followed by the disappearance of phthisical symptoms seems to me to be capable of explanation from the fact that the rising of the clavicle and of the first rib materially assists the retraction of the apex into the chest, and hence is an important factor in the production of a cure.

THE TRUE LINES OF ADVANCE IN ANÆSTHETICS.

BY J. FREDERICK W. SILK, M.D.

IT has always struck me as curious, that more energetic protests have not been raised against the peculiar direction taken by what may be termed the anæsthetic controversy. The true facts of the case are really so simple, that I almost feel called upon to apologise for referring to them at all. These facts I take to be—1. That anæsthetisation is essentially an unnatural process, and, as such, must of necessity involve a certain amount of risk to all who submit themselves to it. 2. That the drugs employed to produce the desired effects are of tolerably well-known strength or anæsthetic power. 3. That, as is the case when drugs are used for other purposes, no two patients respond to the anæsthetic in precisely the same manner. 4. That no two individuals administer in precisely the same way or with precisely the same skill.

There cannot, I should think, be any doubt either as to the truth of these propositions, or as to their forming the proper foundation of any controversy that may arise on the subject. Yet, to judge by the direction taken by the discussions and arguments during the last few years, one would almost imagine that it was assumed at the outset that the process was a purely physiological one, and should entail absolutely no risk ; that little or nothing was known as to the strength of the drugs employed ; and that the only certainties in the whole business were that all patients were alike, and all administrators possessed of equal skill. In fact, that the laws of nature were to be reversed for the special benefit of the anæsthetist.

Upon no other grounds than these can I understand the formation of rival camps of "etherists and chloroformists," or explain the anxiety so freely expressed for the settlement of that much-vexed and altogether illogical question, "Which is the best anæsthetic ?" As long as inquiry is wholly or mainly directed towards the solution of such a point as this, I cannot see that much, if any, permanent good is likely to result. In order to counteract fully such a wide-spread but curiously narrow view of the question, it is quite possible that an elaborate dissertation on the whole subject of anæsthetics might be thought necessary, but it is obvious, of course, that such a dissertation would be quite out of place here. I shall content myself, therefore, with pointing out in as few words as possible what I conceive to be the "true lines of advance" in the subject ; the direction, that is, in which improvement is most possible, and careful observation most likely to yield really useful and practical results.

1. *The preparation of the patient.*—Both from the point of view of the anæsthetist and the operator, this is a most important point ; yet I venture to think that it does not receive the amount of attention that it deserves. We have a somewhat vague and general rule that the anæsthetic should be taken on an empty stomach, and not unfrequently this rule is applied in a curiously arbitrary fashion—to the extent, in fact, of placing the patient on the operating table in a condition as nearly approaching syncope from actual starvation as possible. If it did not lead to any practical results, it would at any rate be interesting to know what, if any, relation existed between the manner in which the anæsthetic was tolerated and the condition of the patient, both with regard to immediate and more remote preparation. In this connection I may perhaps point out that, judging from the usual character of the vomit, the administration of anæsthetics leads to an increased flow of bile ; *primâ facie*,

therefore, one would be inclined to interdict for some hours beforehand any foods which might act as hepatic stimulants, and to limit ourselves to the administration of such substances as can be easily and promptly digested in the stomach itself. I should myself like to know, too, whether, for the same reason, much of the sickness usually ascribed to the anæsthetic may not be referred to the prior administration of some cholagogue purgative; whether, for instance, patients who have had enemata do or do not do better in the matter of sickness than those who have had calomel. Then, again, as to the best time for operating: the text-books are pretty unanimously in favour of the early morning; in London hospitals—as a matter of convenience, I presume, rather than anything else—the afternoon is usually chosen. The question arises, therefore, do patients take the anæsthetic more kindly or better at the one time than the other? Finally, is it or is it not of advantage that the patient should undergo any particular therapeutic treatment before taking an anæsthetic? For instance, some have advised a short course of nux vomica; others have advocated, either as a matter of routine or merely in special cases, the hypodermic injection of morphia. Under this head, too, I would place the administration of stimulants immediately before an operation. This latter is considered by some the most important of all the points connected with the previous preparation of the patient, and there is consequently much difference of opinion expressed; at some institutions, for instance, I believe that the anæsthetist goes round the wards immediately before the operations are commenced, for the express purpose of examining the patients, and, if he thinks necessary, ordering stimulants. Personally, I must confess that I am inclined to doubt whether, under these circumstances, the stimulant has really any beneficial effect, even if it does not actually do harm; but at the same time there is decidedly plenty of room for further observation on the point.

2. *Selection of the anæsthetic.*—It is, I believe, more particularly in the judicious selection of the particular drug to be employed in each individual case that modern practice differs from the somewhat routine methods of former years;

I believe, further, that by studying the results obtained in the use of the several anæsthetics under varying conditions we are much more likely to obtain really useful information than in seeking for a substance which shall be universally applicable, under all circumstances and to every patient. Because the actual number of anæsthetics employed is small, it does not follow that there is proportionately little scope for judgment in their selection. For instance, due regard must be had to the idiosyncrasy of the patient, his age, his previous history, and his present physical condition; and also to the nature and probable duration of the operation. Then, again, the choice is open to us to combine one or more of the drugs, or to alternate their vapours. Finally, we may have to determine whether, in the course of the operation, it may or may not be advisable to substitute one drug for another, or to vary our method of administering. Although, no doubt, we already possess a good deal of information on these several points, and have a few rules to guide us, yet there is ample room for improvement, and a series of careful and extended observations would be very acceptable.

3. *Methods of administration.*—A rag and a bottle no longer constitute the sole armamentaria of the anæsthetist. Simplicity is of course still a very great desideratum, and it by no means follows that he is the best anæsthetist who is armed with the most complicated instruments. On the other hand, more particularly in the administration of nitrous oxide, either alone or in combination with ether, and to a lesser extent in the administration of ether by itself, some form of apparatus is absolutely necessary.

First with regard to nitrous oxide; the fundamental principle (the principle, that is, upon which the majority of anæsthetists are agreed) underlying the administration of this gas is that it shall be given as nearly pure as possible. A secondary principle, or one upon which there is some difference of opinion, I take to be the non-contamination of the gas inhaled with the products of respiration, at any rate in the early stages. It would be unwise to assert that the apparatus at present employed is absolutely perfect, but I think that this may be said with regard to it, that it is more nearly perfect than the apparatus used for the administration of any of the other anæsthetics, and that future improvements will be in details rather than general plan. As an instance of such improvements in details, I would refer to the one suggested by the late Dr. Sheppard, who, by means of a very simple and at the same time ingenious contrivance, overcame one of the most important difficulties connected with the use of gas-bottles in the horizontal rather than the vertical position. In respect to the administration of nitrous oxide and ether in combination, I may perhaps say that the idea is by no manner of means a new one, but, on the other hand, considerable differences of opinion exist both as to its advantages and also as to the best method to be employed in the process.

With regard to ether by itself, the fundamental principle of its administration I take to be the inhalation of not less than 30 per cent. of the vapour ; and as secondary to this the power of regulating the strength of the vapour so inhaled. There are practically two methods by which these results are secured. The ether vapour is either derived from the surface of a known quantity of liquid ether contained in a suitable chamber, or the air to be inspired is made to pass through a saturated sponge. The former plan is the one adopted in Clover's inhalers, the latter is carried into effect in the inhalers that bear the name of Mr. Ormsby. For my own part I prefer to think that the plan of the Clover's inhalers is the best, and scientifically the most accurate ; at the same time, however, I am prepared to admit that these inhalers are open to many objections, and it is precisely in this direction that I think that there is scope for improvement. For instance, they are rather clumsy, complicated, and costly ; then, again, they are uncleanly, or perhaps I should say that they cannot be easily cleaned, and in these days especially this is a grievous fault. Here, then, is an opportunity for anyone possessed of the necessary mechanical ingenuity and skill.

With regard to chloroform, matters are on a somewhat different footing. The fundamental principle, I should say, was that as little as possible of the drug should be given, and certainly not more than 4 per cent. From this point authorities diverge widely, some advocating what may be termed ultra-simplicity, *e.g.* a piece of lint, &c.,—and those who employ this method urge that, even if it necessitates more trouble and greater care on the part of the administrator, at any rate it is to be preferred to the over-confidence sometimes induced by more mechanical methods; on the other hand, a good many anæsthetists employ what is known as Junker's inhaler, with the principles and action of which, as well as with its advantages and disadvantages, I shall presume that all Guy's men are perfectly familiar. It is not my purpose to discuss the relative merits of these two methods, but I shall content myself with saying that neither is perfect, and that there is ample room for improvement.

4. *The phenomena of anæsthetisation.*—One cannot of course pretend that the phenomena resulting from the administration of anæsthetics are either so numerous or so varied as those observed in many other departments of practice. They have, however, been thought to be sufficiently important and interesting to call forth some of the best and most elaborate work of such skilled investigators as Dr. Lauder Brunton, Professor Wood, Professor MacWilliam, and many others; the subject cannot, therefore, be considered as being altogether unworthy of our attention.

For some time the point at issue was as to whether the ultimate cause of death in fatal cases was due to failure of the respiratory centres or to failure of the heart; the consensus of opinion now inclines to respiratory rather than cardiac failure in these cases. But a point of far greater importance to the practical anæsthetist, and concerning which we still stand in need of reliable clinical information, is as to the exact significance of the alterations observed in the circulatory and respiratory systems during the actual process of anæsthetisation.

Then, again, with regard to the pupil changes; as is so well known, these are apparently both numerous and very irregular. At present, with a few exceptions, we neither know for certain what a particular condition of the pupil may mean, nor can we say whether it may or may not foretell anything; yet surely such phenomena cannot be altogether without significance.

5. *The treatment of emergencies.*—Personally I am one of those who believe that fussy anæsthetisation, if I may use the phrase, is as bad in its way as "meddlesome midwifery," and that no small number of lives have been lost from over-active interference on the part of the administrator or his assistants. Whether this actually is so or not, it would be very desirable to have some reliable observations on the point, and to have some definite statements, not only as to what we should do, but also as to what we should not do in cases of difficulty.

6. *The after-effects and after-treatment.*—Of course we all know that many evils are laid at the door of the anæsthetist which do not rightly belong to him; but apart from this it would be of great interest, at least, to know definitely what are the exact effects of the anæsthetic on the secretion of urine in health and disease, upon the excretion of sugar in diabetes, &c. Then, again, but little is known, and I think that more might be known with advantage, as to the treatment of some of the distressing symptoms which so frequently follow the use of an anæsthetic; for instance, the sickness, the depression, &c.

I think that I have now said enough to show that the field of anæsthetics is by no means likely to prove unfruitful, and how it might be cultivated much more profitably than in attempting to read sphinx-like riddles or answer useless conundrums.

SOME POINTS

IN THE

MORBID ANATOMY OF LEPROSY.

By BEAVEN RAKE, M.D.

(Communicated by the EDITORS.)

During the past eight years the writer has had opportunities of making 109 autopsies on lepers in the Trinidad Leper Asylum, and it has therefore seemed worth while to put the notes together, and to endeavour to draw some conclusions from them. It is right to state at the outset that many of these observations have already been published, but a complete summary of all the cases has not appeared before. Several of the specimens are now in the Museum of Guy's Hospital. In estimating the value of any deductions, allowance must of course be made for the limited number of these observations. They, however, probably represent the largest number of autopsies on lepers yet made in any one place within a comparatively short space of time.

A detailed statement of the results of post-mortem examination will be found at the end of the paper, and before examining some of the more interesting questions suggested by the table it may be well to consider what general information can be obtained from this mass of facts and figures.

It may be remarked that these cases are recorded without selection, and exactly in the order in which they occurred, as appears from the dates in the table.

It will be seen that the three forms of leprosy are almost equally represented, the numbers being—

Tuberculated . . .	36
Anæsthetic . . .	42
Mixed . . .	31
Total . .	109

The mixed variety has long been recognised as a connecting link between the other two forms of leprosy, and it is a noteworthy fact that a case of tuberculated leprosy, if the patient lives long enough, generally develops more or less anæsthesia, and the nerves after death are frequently found to be infiltrated with leprous growth. The converse condition, the development of cutaneous tubercles in a case of pure anæsthetic leprosy, the writer has never seen. Leloir,[1] however, mentions three such cases.

Recent dissections by Gerlach[2] show that in anæsthetic leprosy the primary growth takes place in the skin around the peripheral ends of the nerves, extending afterwards up the branches to the main trunks. These observations, if confirmed, will bring the two forms of leprosy much closer together, for the mode of onset will thus become the same in both. A possible explanation of the more frequent transition of a tuberculated than of an anæsthetic into a mixed case is also afforded by Gerlach's researches. In tuberculated leprosy large tracts of skin are infiltrated with new growth containing quantities of bacilli, whereas in anæsthetic leprosy the new growth occurs in limited areas around the peripheral nerve-endings, and soon travels up the nerves, leaving the skin practically free from bacilli, for it is well known that these are seldom or never found in the skin in advanced cases of anæsthetic leprosy. Now it is obvious that the risk which the nerves passing through the infiltrated areas in tuberculated leprosy run of becoming secondarily involved by extension from these areas is much greater than the risk which is incurred in anæsthetic leprosy, of the skin becoming infiltrated by a growth which, after the first onset, is practically confined to the nerves. The condition of the skin as to bacilli in extremely early cases of anæsthetic leprosy will be a matter for further research. Meanwhile it may be accepted as a fairly accurate statement, that cases of tuberculated leprosy are those which have not lived long enough to become mixed.

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[1] 'Traité théorique et pratique de la lèpre,' p. 209.

[2] 'Untersuchungen über die Unabhängigkeit der Bildung anästhetischer Hautflecke von der Erkrankung zugehöriger Nerven bei der Lepra anästhetica,' Dorpat, 1890.

The next point which demands attention is the average duration of the disease in each of the three forms. The number of years during which the patient had been a leper was only ascertained in ninety-two of the hundred and nine cases. Particulars are given in the following table.

Form of leprosy.	Number of cases.	Average duration of disease in years.
Tuberculated	30	6·4
Anæsthetic	32	10·8
Mixed	30	9·5
Total	92	9

It thus appears that the duration of life in tuberculated leprosy is about two thirds that in mixed leprosy, while anæsthetic leprosy has a longer course than either of the other two. The reason of this is not apparent, for, as will be seen in subsequent tables, visceral lesions are almost if not quite as common in anæsthetic as in tuberculated leprosy.

In the next table the expectation of life in leprosy in Trinidad is compared with that in Demerara and Norway.

Form of leprosy.	Average duration of disease in years.		
	Trinidad.	Demerara.	Norway.
Tuberculated	6·4	8·5	9·5
Anæsthetic	10·8	15	18·5
Mixed	9·5	6	No return.

This table shows that the average duration of tuberculated and anæsthetic leprosy in Demerara and Norway, as calculated by Hillis and Danielssen is greater than that in

Trinidad. Mixed leprosy appears to run a shorter course in Demerara, while in Norway no returns are given. The above-mentioned authors, however, exclude such diseases as phthisis and kidney disease, which they consider are intercurrent, and therefore unfairly reduce the average duration of leprosy as a disease considered apart. But, as will be shown further on, it is by no means certain that such diseases as those mentioned above are not dependent directly or indirectly on leprous lesions; and even if they are intercurrent, the expectation of life in leprosy is not affected by including them, as appears from the following table.

Form of leprosy.	Average duration of disease in years.		
	All cases of leprosy.	Leprosy with kidney disease.	Leprosy with tuberculosis.
Tuberculated .	6·4	7	5
Anæsthetic .	10·8	17	13
Mixed . .	9·5	5	6
Total .	9	10	8

Omitting decimals, the average duration of all cases of leprosy is found to be nine years, while that in leprosy with kidney disease and leprosy with tuberculosis is ten years and eight years respectively. If these two complications are taken together, the average duration is found to be nine years, exactly the same as in all cases of leprosy.

The next table shows the average age at death in the three forms of leprosy. The ages obtained can, however, only be regarded as approximate, for members of dark-skinned races, as a rule, are very uncertain as to their ages, and a more or less rough estimate has often to be made.

Form of leprosy.	Age at death.	Age at onset.
Tuberculated . .	24	18
Anæsthetic . .	44	33
Mixed . . .	40	30
Total .	36	27

Decimals have again been omitted in this table. As far as the figures can be trusted, they show that tuberculated leprosy appears much earlier in life than either of the other forms. But as it has already been shown that mixed leprosy is usually a sequel of the pure tuberculated variety, it ought to begin at about the same age. It is, therefore, probable that the last figures are not accurate.

The birthplaces of the 109 patients are as follows :

Trinidad	59
India	30
Africa	6
China	2
Barbados	2
Germany	2
Demerara	1
France	1
Grenada	1
Madeira	1
Martinique	1
Nevis	1
Tobago	1
Born at sea	1
Total	109

It is worthy of remark that only three Europeans occur in the list, two of these being born in Germany and one in France. It must, however, be remembered that Europeans belonging to the lower classes are not numerous in Trinidad. As would naturally be expected, Trinidad supplies more than half of the total number, while a very considerable proportion is represented by Indian immigrants. The latter, before they are employed on the estates, are subjected to three medical examinations, one in Calcutta and two in Trinidad, and it is rarely that leprosy appears before the expiration of their five years' indenture. From these facts two inferences are possible ; either that the usual incubation of leprosy in them is more than five years, or that they acquire the disease in Trinidad. Judging from recorded cases in which leprosy has developed in persons who have removed from an endemic area to a country free from indigenous leprosy, the latter seems the more probable hypothesis.

Lastly, reference must be made to the lesions found after death in the cases of leprosy under consideration. These are given in the following summary.

Chief Lesions found after Death.

Kidney disease	35	Cardiac hypertrophy	2
Tuberculosis	33	Abscess of liver	2
Ulceration and gangrene	12	Emphysema	1
Pyæmia	8	Addison's disease	1
Obstruction of larynx	7	Malarial fever	1
Ankylostomiasis	7	Sunstroke	1
Abscess and hæmorrhage	4	Facial carbuncle	1
Pneumonia	4	Cystitis	1
Cirrhosis of liver	4	Cerebral thrombosis	1
Gangrene of lungs	3	Hydronephrosis	1
Atheroma	3	Sacro-iliac disease	1
Dysentery	3	Cancer of uterus	1
Lardaceous degeneration	3	Strangulated hernia	1
Pleurisy	3	Hip-joint disease	1
Syphilis	2	Aortic aneurism	1
Pericarditis	2	Total	150

In many cases several different lesions were found in the same body, hence the total is considerably in excess of the number of subjects examined. It is at once evident, on looking at the table, that a very large proportion of the lesions is the result either of some form of kidney disease or of tuberculosis. As will be pointed out presently, the latter term is only used provisionally, and it may be necessary to change it at no distant date.

For the purposes of this paper the lesions mentioned in the above list may be divided into three groups:

Kidney disease	35
Tuberculosis	33
Other lesions	82
Total	150

It is proposed to examine these groups separately, and to conclude with a few remarks on the distribution of leprosy bacilli in the tissues of the body.

Kidney Disease.

Kidney disease has long been known to be common in leprosy. Of the 109 bodies examined, 35, or 32 per cent.

showed some form of nephritis. Other renal changes, such as lardaceous degeneration, pyæmic infarction, and syphilitic and tubercular deposit, have purposely been omitted from this calculation. Sometimes, however, these changes have occurred in addition to the nephritis.

In order to determine whether kidney lesions are commoner in lepers than in persons free from leprosy, the records of deaths in the Colonial Hospital, Port of Spain, were examined, and it was found that 7½ per cent. of the cases showed some form of kidney disease. Thus the percentage of cases of nephritis in the Leper Asylum appears to be more than four times that of similar lesions in the General Hospital. An allowance must be made for the fact that in the latter institution some of the kidneys probably escaped examination; but even if the figures are doubled or trebled, the percentage of kidney disease in the asylum will still be greatly in excess of that in the hospital.

Various statements have been made as to the nature of the kidney lesions in leprosy. Danielssen and Boeck[1] say of tuberculated leprosy, "Les reins sont presque constamment plus ou moins attaqués si la maladie a duré longtemps, si même les autres organes internes sont demeurés tout à fait intacts. La capsule des reins est souvent revêtue de petits tubercules, la substance rénale est aussi plus ou moins affectée. Nous ne doutons aucunement que chacun ne soit porté a reconnaître dans ces altérations celle de la néphrite albumineuse, si parfaitement décrite et représentée par Rayer." Of anæsthetic leprosy they remark, "Les altérations sont les mêmes que celles constatées par nous sous la forme tuberculeuse, seulement nous avons remarqué ici deux fois une grande quantité de cystides formés surtout dans la substance rénale."

It is quite possible that the tubercles described by the above authors in tuberculated leprosy are identical with those presently to be discussed under the head of tuberculosis. The writer has also observed cysts in the kidney in several cases of leprosy, but these did not appear to differ from those found in non-leprous persons.

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[1] 'Traité de la Spedalskhed,' pp. 226, 289.

Bidenkap[1] regards the kidney changes as specific, and says that albuminuria is not uncommon in tuberculated leprosy.

Hillis[2] says albuminous nephritis causes 22½ per cent. of the deaths in tuberculated leprosy. In anæsthetic leprosy he mentions dropsy as a cause of death in 11 per cent. of the cases, but says that it is not dependent on nephritis.

Cornil and Babes[3] do not seem to recognise the ordinary forms of chronic nephritis in leprosy, but describe albuminous nephritis as coming on together with lardaceous changes in other viscera as the result of ulcerations of the skin and mucous membranes. They also state that the *Bacillus lepræ* may invade the kidneys, together with all the tissues of the body, without producing any naked-eye change.

Leloir[4] states that Hansen has never found leprous changes in the kidneys.

The following table shows the number and varieties of kidney lesions occurring in the thirty-five cases of leprosy already mentioned.

Form of leprosy.	Form of kidney disease.				
	Acute nephritis.	Large white kidney.	Mixed kidney.	Contracted kidney.	Total.
Tuberculated .	1	9	3	2	15
Anæsthetic . .	—	3	8	1	12
Mixed . .	—	2	4	2	8
Total . .	1	14	15	5	35

The case of acute nephritis scarcely comes within the scope of the present inquiry, for the left kidney was congenitally atrophied, weighing only half a drachm, and probably the acute attack was in a great measure caused by the almost double work which devolved on the right kidney.

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[1] 'Lectures on Lepra,' p. 47.

[2] 'Leprosy in British Guiana,' pp. 39, 111.

[3] 'Les Bactéries,' p. 769.

[4] Op. cit., p. 256.

If this case is subtracted it will be seen that the total number of cases of kidney disease is almost the same in the tuberculated and anæsthetic leprosy, while in mixed leprosy the kidney lesions are less numerous. The different forms of chronic kidney lesion are almost equally distributed in the different varieties of leprosy, with the exception of the large white kidney, which shows a preponderance in cases of tuberculated leprosy, and of the mixed kidney, which shows an almost equal preponderance in cases of anæsthetic leprosy.

The next table shows the average duration of the different forms of leprosy when associated with the varieties of kidney disease already mentioned.

Form of leprosy.	Average duration of leprosy in different forms of kidney disease.				
	Acute nephritis.	Large white kidney.	Mixed kidney.	Contracted kidney.	Total.
Tuberculated .	7	7	7	8	7
Anæsthetic . .	—	19	14	—	17
Mixed . .	—	4	6	—	5
Total . .	7	10	9	8	10

As will be seen on reference to the large table at the end of this paper, kidney disease was often associated with lesions of other viscera. Hence it is probable that in some cases nephritis was not alone responsible for shortening life, and the above figures are not strictly accurate. In eight cases kidney disease co-existed with tuberculosis. All these have been eliminated from this and the corresponding table in the next section, for the results obtained both in kidney disease and tuberculosis would thus have been considerably vitiated. As it is, the figures, though not strictly true, may be regarded as approximate.

The most noticeable point in this last table is the much longer duration of life in anæsthetic leprosy associated with kidney disease than in either of the other two forms of leprosy when occurring in conjunction with renal changes. Two patients with large white kidneys had suffered from anæsthetic leprosy for twenty-three and fifteen years respectively, while four patients with mixed kidney had been anæsthetic lepers for thirty-four, twenty, seventeen, and thirteen years respectively. This is possibly to be explained by the different degrees in which the sweat-glands are involved in the three varieties of leprosy. The cutaneous changes are more rapid and extensive in tuberculated and mixed than in anæsthetic leprosy. In the first two forms a sudden eruption of tubercles may damage a large number of sudoriparous glands, and so make a sudden demand on the secreting structure of the kidney. In anæsthetic leprosy, on the other hand, the changes in the sweat-glands are more gradual and often of less severity, so that the strain thrown on the kidneys is less.

The kidneys were examined microscopically in seventy-seven of the hundred and nine cases, and in only seven instances were leprosy bacilli found, twice in tuberculated and five times in mixed leprosy. It is an interesting fact that in all these seven cases the kidneys appeared healthy to the naked eye. This agrees with the statement of Cornil and Babes already quoted.

In none of the thirty-five cases of kidney disease were leprosy bacilli found. It may, therefore, be considered highly probable that the forms of nephritis which occur in leprosy are, at least in the majority of instances, not due to leprous growth in the kidneys, but are the result of interference with the functions of the skin. This hypothesis is supported by the fact already referred to of the longer duration of cases of kidney disease associated with anæsthetic leprosy.

Tuberculosis.

Visceral tuberculosis has been described in leprosy by various writers. Of the 109 subjects examined, tuberculosis of one or more serous membranes or viscera was found in 33, or 30 per cent. The records of the Colonial Hospital, already referred to, show a percentage of eighteen and a half deaths from tuberculosis. It must be remembered that cases of phthisis are excluded, if possible, from a general hospital, though this objection perhaps has not the same force in the tropics, where patients are often taken in to prevent their dying in the streets. Even if the objection be admitted, it will probably still be safe to infer that deaths from tuberculosis, especially of the lungs, are commoner in lepers than in the ordinary population.

A question naturally arises whether these lesions are due to invasion by the bacillus of leprosy or the bacillus of tubercle. The answer is not as easy as at first sight appears. Danielssen[1] says, "Il est difficile d'admettre que deux germes différentes, ayant pénétré à peu près à la même époque dans de sang, aient pu produire, chacun de son côté, des maladies telles que la lèpre et la tuberculose. Il me parait beaucoup plus probable que la même cause d'irritation (bacilles de la lèpre) qui produit dans la peau des éruptions, dans le tissu conjonctif des viscères des néoformations, puisse donner naissance, dans d'autres tissus de l'organism, à la tuberculose."

He calls attention to the fact that the disappearance of cutaneous tubercles in a case of leprosy is not infrequently followed soon after by the development of visceral tuberculosis, and he raises the question whether the bacillus of leprosy and that of tuberculosis are identical. He is led to make this suggestion by the extremely unsatisfactory distinction between the two bacilli. With regard to the alleged difference in size, he states that bacilli from cases of leprosy in Spain are larger than leprosy bacilli from Norway.

Arning[2] writes as follows:—"In all advanced tubercular cases I was struck with the extreme frequency of grave changes in the larger viscera, more especially the lungs, liver, spleen, and bowels. These organs presented an aspect quite new to me, and closer examination of their tissues has enabled me to prove that we have been mistaken in attributing deaths of lepers to intercurrent pneumonia, tubercular phthisis, and dysentery, which were simulated by the clinical symptoms. The ulcerations of the bowels and the breaking down of lung tissue are due to leprous infiltrations, and we shall have to modify our opinions of leprosy being mainly a disease of the cutis and peripheral

[1] "Contribution à l'étude de la lèpre," 'Archives Romaines,' Jan., 1889.

[2] 'Report to Hawaiian Board of Health,' 1886, Appendix, p. xli.

nerves, and introduce terms such as phthisis leprosa and enteritis leprosa."

Hansen,[1] on the other hand, regards the changes found in the lungs, intestines, bronchi, brain, and spinal cord of lepers as tubercular, not leprous.

Damaschino[2] states that he has succeeded in producing tuberculosis in guinea-pigs by inoculating them with fragments of phthisical lung from lepers. He is thus led to regard the phthisis of leprosy as produced by the *Bacillus tuberculosis,* but he says that he has also recognised the *Bacillus lepræ* in the lungs in these cases. This appears to be a somewhat bold diagnosis.

The above quotations show what very different statements have been made as to the nature of visceral tuberculosis in leprosy. Before proceeding to examine the evidence obtainable from the Trinidad Asylum it will be well to give two short tables prepared with reference to the cases under consideration.

The first table shows the various viscera and serous membranes affected, and the distribution of these lesions in the different forms of leprosy.

Form of leprosy.	Viscera affected.										Total.
	Lungs.	Lungs and peritoneum.	Pleura.	Lungs and liver.	Lungs, liver, kidney, spleen, pleura, omentum, and peritoneum.	Omentum and peritoneum.	Lungs, liver, and spleen.	Lungs and spleen.	Lungs, spleen, and kidneys.	Liver, kidneys, spleen and peritoneum.	
Tuberculated .	5	—	1	1	—	—	2	1	1	1	12
Anæsthetic .	8	1	—	—	—	—	1	1	1	—	12
Mixed .	4	1	—	1	1	1	—	—	1	—	9
Total .	17	2	1	2	1	1	3	2	3	1	33

The tubercles observed in the viscera had the naked-eye appearances presented by tubercles in non-leprous subjects. As is shown in the large table at the end of this paper, destruction of lung tissue was in many cases very rapid, the lungs being speedily converted into bags of pus. It will be noticed that in only three cases did the lungs escape. In one of these the pleura only was affected, in another the omentum and peritoneum, and in another the liver, kidneys, spleen, and peritoneum. In those cases in which the lungs alone were affected the leprosy was tuberculated in five instances, anæsthetic in eight, and mixed in four. Taking, however, the total cases of visceral infection, the three forms of leprosy are almost equally represented, twelve cases being tuberculated, twelve anæsthetic, and nine mixed. It may, therefore, be concluded that visceral tuberculosis is quite as common in anæsthetic leprosy as in either of the other forms.

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[1] Leloir, op. cit., p. 256.

[2] 'Arch. de méd. et d'anat. Path.,' Paris, 1891, p. 213.

The next table shows the average duration of the different forms of leprosy in the above varieties of visceral tuberculosis.

Form of leprosy.	Average duration of leprosy in different varieties of visceral tuberculosis.										Total.
	Lungs.	Lungs and peritoneum.	Pleura.	Lungs and liver.	Lungs, liver, kidney, spleen, pleura, omentum, and peritoneum.	Omentum and peritoneum.	Lungs, liver, and spleen.	Lungs and spleen.	Lungs, spleen, and kidneys.	Liver, kidneys, spleen and peritoneum.	
Tuberculated .	7	—	—	5	—	—	5	2	—	—	5
Anæsthetic .	7	—	—	—	—	—	—	18	—	—	13
Mixed . .	11	3	—	5	—	—	—	—	—	—	6
Total .	8	3	—	5	—	—	5	10	—	—	8

In many of the cases the duration of the leprosy could not be ascertained. The above table is therefore very fragmentary, but the general results obtainable from it are almost the same as in cases of leprosy with kidney disease, and not very different from the average durations in all cases of leprosy.

Seeing that the distinctions between the bacillus of leprosy and that of tubercle are so very unsatisfactory, it may be well to consider here the other arguments for and against the leprous nature of visceral tuberculosis.

In the first place, all attempts to cultivate bacilli from fragments of phthisical lung or tuberculous viscera failed in the Trinidad Asylum. This, so far as negative evidence is of value, supports the theory of a leprous origin, for it is by no means certain that the *Bacillus lepræ* has been cultivated. The results of Bordoni Uffreduzzi,[1] Gianturco,[2] and Campana[3] have not yet been confirmed.

Secondly, attempts to infect fowls by feeding with fragments of phthisical lung failed in Trinidad. Other results, however, were obtained in guinea-pigs by Damaschino, as was already pointed out.

Thirdly, in several cases of tubercular and mixed leprosy in which cutaneous tubercles rapidly diminished towards the end, numerous miliary tubercles were found in the viscera after death, as described by Danielssen. These appearances were especially marked in Cases 58, 71, 75, 86, 90, 91. It has, however, been already shown that visceral tuberculosis is quite as common in anæsthetic leprosy as in either of the other two forms. Of the twelve cases of visceral tuberculosis in anæsthetic lepers, four were of the miliary variety indicating recent infection. In these cases there could not have been absorption of cutaneous tubercles.

From the above observations and experiments it seems, therefore, that the evidence at present is almost as much in favour of tuberculosis as of leprosy as the cause of the visceral lesions above referred to.

Other Lesions.

Ulceration and gangrene were present to some extent in very many of the cases examined. Indeed, it is seldom that a leper passes through life without suffering in this way at one time or another. Ulceration is very apt to spread rapidly shortly before death, and to become complicated with gangrene. These lesions were present to a marked degree in twelve cases only. The amount of gangrene in the Trinidad Asylum is much less than it used to be. This diminution is very probably in great part due to the practice now carried out of early and free incision down to the bone in cases of sinus or ulcer. Amputation for gangrene has also been followed by good results in several cases.

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[1] 'Zeitschrift für Hygiene,' Band iii, p. 178.

[2] 'Gior. della Assoz. Nap. di Med. et Nat.,' 1890.

[3] 'Riforma Med.,' No. 14, 1891.

Pyæmia was found in eight cases. In one case it followed amputation through the thigh for elephantiasis, from which the patient suffered in addition to leprosy. In another case it occurred as a sequel of facial carbuncle, the necropsy revealing thrombosis of the ophthalmic veins and pus in the cavernous sinus. In two cases absorption took place from gangrene of the legs, while in the four remaining cases pyæmia followed sloughing ulcers of the cæcum, suppurating cervical glands, sacro-iliac disease, and ulceration of the larynx respectively. It is remarkable that pyæmia does not occur more frequently in leprosy.

In seven cases death occurred from obstruction of the larynx caused by leprous changes. In one patient, who afterwards died of phthisis, life was prolonged for four months by tracheotomy, and in Norway[1] several years of life have often been gained by this operation. As a matter of fact it would be safer always to perform tracheotomy as soon as the larynx is distinctly involved, for symptoms of obstruction often come on very rapidly in lepers, and death from asphyxia may occur before surgical assistance can be obtained.

The ankylostoma duodenale was present in greater or less quantity in seven instances. In Case 108 death was traceable to the intense anæmia following the intestinal hæmorrhages produced by the parasite. The liver yielded, on analysis by Mr. Carmody, Government Analyst, ·26 per cent. of iron, while the spleen gave 3·28 per cent. In the other cases the presence of ankylostomata no doubt contributed to the fatal result. These worms are very common in Trinidad, and are found chiefly amongst the Indian immigrants. They give rise during life to symptoms indistinguishable from those of pernicious anæmia.

In four cases death was due to abscess or hæmorrhage or both. In three of these cases hæmorrhage came on after amputation for abscess or gangrene, and the patients, already

[1] Abraham, 'Epidem. Soc. Trans.,' vol. viii, p. 135.

weakened by suppuration, succumbed. Generally speaking, the blood of lepers clots very rapidly, and healing takes place quite as easily, if not more so than in non-leprous subjects. Case 44 is interesting in this respect. A thoracic aneurism was found which had undergone spontaneous cure. The specimen is now in Guy's Hospital Museum. The blood of lepers has been examined by Danielssen and Boeck,[1] Hillairet,[2] and the writer,[3] and their results, though probably not strictly accurate, tend to show that the percentage of fibrin in the blood of lepers is greater than in that of ordinary people.

Pneumonia was found in four cases, and presented no points of special interest. In two cases it was associated with pyæmia, in a third it occurred in a patient broken down by alcoholic excess, while in the fourth case it followed influenza.

Cirrhosis of the liver occurred in four lepers,—once in a patient who was also the subject of large white kidney, once with atheroma of the orifices of the heart, once in association with granular kidney and tubercle of the lungs and peritoneum, and once alone.

Gangrene of the lung was found three times. In the first case the patient was a confirmed alcoholic; the second patient suffered from kidney disease; the third case was in an old patient who showed no other visceral lesion beyond fatty degeneration of the liver and kidneys.

Atheroma was markedly present in three subjects. In the first case there were sharp calcareous masses at each of the four valvular orifices of the heart, but these were largest above the mitral and aortic valves. In the next case there was atheroma of the circle of Willis, giving rise to thrombosis. In the third case there were patches of atheroma on the aorta, mitral valve, and one of the aortic valves. The mitral orifice was contracted, and the heart was considerably hypertrophied, weighing 13 oz. Atheroma was, of course, present in a less degree in other instances, but in these three it was more or less concerned in causing death.

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[1] 'Traité de la Spedalskhed,' pp. 238, 296.

[2] 'Ann. de Dermat. et Syphilog.,' tome v, No. 3.

[3] 'Lancet,' January 9th, 1892.

Dysentery materially contributed to the fatal result in three cases. In one it occurred alone in an old patient, and in the other two it was associated with abscess of the liver and kidney disease respectively.

Lardaceous degeneration was found three times,—once in association with phthisis, once with kidney disease, and once with superficial ulceration of the trunk and extremities.

Though more or less pleuritic adhesion was found in nearly every subject examined, pleurisy to a marked degree was only seen in three cases. In these it occurred in conjunction with abscess of the liver, tuberculosis of the lungs, and kidney disease and acute pericarditis respectively.

Evidences of syphilis existed in several cases, but in Cases 4 and 32 they were most marked, being represented in the first case by fibrous testes, gummata of the liver, spleen, and kidneys, and a gummatous mass round the seventh pair of nerves at their exit from the skull, and in the second case by fibrous testes and destruction of the epiglottis and bones of the nose.

Pericarditis occurred in two patients, once in association with kidney disease and once with tuberculosis. In the first case there were several ounces of fluid in the pericardium, and some flakes of lymph on the walls of the heart. In the other case the disease was evidently more chronic, for the pericardium was filled with layers of partly organised lymph, altogether about three quarters of an inch thick.

Cardiac hypertrophy was found in two cases. In one there was atheroma of the valves, but in the other no sufficient cause was found, the chief conditions recognised being ulceration of the extremities and of the epiglottis and vocal cords.

Abscess of the liver also occurred twice. In both patients there were multiple abscesses. In the first case there were a few small abscesses near the portal fissure, the largest only three quarters of an inch in diameter; while in the second case there were five large abscesses, containing from two to three pints of pus. The first patient was the subject of ulceration and gangrene; the second suffered also from dysentery.

Disease of the adrenals was found in one case associated with asthenia and numerous pigmentary changes. The writer has not found any record of the occurrence of this disease in dark-skinned races. There were evidences of syphilis in the case referred to, but beyond the adrenal disease no lesions were found which would have been sufficient to cause death.

Sunstroke was accountable for death in one case. This is a very rare affection in Trinidad in spite of its tropical climate. The patient, who suddenly fell down unconscious while sitting out of doors, died exactly twenty-four hours after the onset of the symptoms, and a necropsy two hours after death showed no lesion beyond congestion of some of the viscera. The temperature in the centre of the liver was found to be 106°, while in the spleen it was 103·2°, and in the right and left pleural cavities 102·2° and 101·4° respectively.

Apparently uncomplicated malarial fever caused death in one patient. This is a rare occurrence at the asylum, for though malarial fever is very common among the inmates, death seldom results from this disease alone.

Facial carbuncle occurred in a leper who was subject to maniacal attacks, and who used to put his head in the oven to get rid of supposed evil spirits. Insanity is not uncommon amongst lepers, but no leprous changes have ever been found within the cranium by the writer. Chassiotis,[1] however, says that he has detected leprosy bacilli in the spinal cord.

Cystitis was found in one case as the result of the introduction of a straw four and a half inches long into the urethra by the patient himself. This straw had evidently been some time at the neck of the bladder, and was thickly coated with phosphates. The specimen is now in Guy's Hospital Museum.

Death was caused once by thrombosis, resulting from atheroma of the circle of Willis in an old patient.

Of the remaining lesions in the list, namely, emphysema, hydronephrosis, sacro-iliac disease, cancer of the uterus, strangulated hernia, and traumatic hip-joint disease, single examples occurred. They are sufficiently described in the table at the end of this paper, and do not call for special comment.

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[1] 'Monatsschrifte für prakt. Dermat.,' Hamburg, 1887.

On looking through the list of lesions which has now been considered, it is worthy of note that, with the exception of ulceration, gangrene, abscess, obstruction of the larynx from leprous deposit, and some cases of pyæmia, namely, those in which absorption from gangrenous ulcers of the skin or larynx appears to have taken place, none of the morbid changes can with certainty be directly traced to leprosy. In other words, reckoning half the cases of pyæmia as due to leprous absorption, only twenty-seven lesions out of one hundred and fifty can be regarded as undoubtedly traceable to leprous changes. Many of the lesions at the end of the list are obviously quite independent, while the ætiology of others, as was shown earlier in this paper, is still uncertain.

The Distribution of the Leprosy bacillus.

Lastly, a few words may be said as to the distribution of the leprosy bacillus in the tissues. The result of microscopic examination in one hundred out of the hundred and nine cases is given in the next table.

Besides the material here referred to, several other viscera and tissues were examined with negative result. These included brain, pons, spinal cord, anterior cervical and dorsal roots, ganglion of vagus, ganglion of aortic plexus, atheromatous cerebral artery, mediastinal, bronchial, and renal glands, tubercles of mesentery, pleura, omentum, intestine, and diaphragm, ante-mortem clot and infarcts, medulla of bone, adrenal bodies, ovarian cyst, cancer of uterus, heart muscle, anæsthetic skin, and discharge from ulcers and abscesses. The number of these additional examinations amounted to fifty. Adding this to the three hundred and eighty-one negative examinations recorded in the table, a total of four hundred and thirty-one instances in which bacilli were not found is obtained. If to this again be added the hundred and fifty observations which showed leprosy bacilli, it will be seen that these

Table of Distribution of Leprosy Bacilli in One Hundred Cases of Leprosy examined after Death.

Material examined.	Bacilli.																		
	Numerous.				Generally diffused.				Few.				Total number of cases in which bacilli were found.	None.				Total number of cases examined.	Percentage of cases in which bacilli were found.
	Tub.	Anæsth.	Mixed.	Total.	Tub.	Anæsth.	Mixed.	Total.	Tub.	Anæsth.	Mixed	Total.		Tub.	Anæsth.	Mixed.	Total.		
Femoral gland . . .	10	...	6	16	3	2	3	8	4	2	6	12	36	13	23	14	50	86	42
Liver	2	...	3	5	2	1	2	5	5	1	6	12	22	17	32	13	62	84	26
Larynx	3	...	8	11	2	1	2	5	...	...	2	2	18	3	...	2	5	23	78
Median nerve . . .	2	3	1	6	...	2	1	3	2	3	3	8	17	17	23	22	62	79	21
Spleen	2	...	5	7	2	1	...	3	3	...	3	6	16	15	30	13	58	74	21
Tubercle of skin . .	2	...	2	4	2	...	...	2	1	...	2	3	9	2	...	1	3	12	75
Testis	2	...	2	4	...	...	1	1	1	...	1	2	7	7	12	11	30	37	19
Kidney	1	...	1	2	...	...	1	1	1	...	3	4	7	24	31	15	70	77	9
Lung	1	...	1	2	1	...	...	1	...	1	3	4	7	10	15	6	31	38	18
Epiglottis. . . .	...	...	...	...	2	...	...	2	...	...	...	...	2	...	...	...	...	2	
Mesenteric gland . .	...	...	...	...	...	...	...	...	1	...	1	2	2	1	...	...	1	3	
Intestine	...	...	...	...	1	...	...	1	...	...	...	...	1	1	2	...	3	4	
Sciatic nerve . . .	...	...	...	...	...	...	...	...	...	...	1	1	1	1	2	1	4	5	
Ulnar nerve . . .	...	...	...	...	...	...	...	...	1	...	...	1	1	1	...	...	1	2	
Superior cervical ganglion	...	1	...	1	...	...	...	...	...	...	...	...	1	...	...	...	...	1	
Tongue	...	...	...	...	...	...	...	...	...	...	1	1	1	1	...	...	1	2	
Solitary follicle . .	...	...	...	...	...	...	...	...	1	...	...	1	1	...	...	...	...	1	
Lumbar gland . . .	...	...	...	...	...	...	...	...	1	...	...	1	1	...	...	...	...	1	
Total . . .	25	4	29	58	15	7	10	32	21	7	32	60	150	113	170	98	381	531	

were found in one hundred and fifty out of five hundred and eighty-one observations, or rather more than 25 per cent.

It must be clearly understood that these results are only of the roughest description. To form any accurate idea of the exemption or not from leprosy bacilli of a tissue or viscus, many examinations of different parts of the structure would have to be made, for it is well known that the distribution of bacilli in a tissue or viscus may be very local. The figures given above only represent the results of routine examinations of fresh tissues made immediately after the necropsies, and would be more correctly described as referring to the tissues and viscera in which bacilli are most easily found.

It may perhaps seem remarkable that so few examinations of cutaneous tubercle appear in the list, but this material is obtained so much better during life that most of the specimens examined were taken from living patients. The results of such examinations have, therefore, no place in this table, which concerns only dead subjects.

It will not be fair to calculate percentages for more than the first nine tissues and viscera in which bacilli were found, for so few specimens of those in the second half of the list were examined that the percentages obtained would be valueless. It will be seen that the highest percentage of successful examinations was in the larynx, and was seventy-eight. Omitting decimals, the other percentages were cutaneous tubercles seventy-five, femoral glands forty-two, liver twenty-six, spleen twenty-one, median nerves twenty-one, testes nineteen, lungs eighteen, and kidneys nine. It is a striking fact that bacilli are very much less common in the kidneys than in any other of the viscera which have been frequently examined. In the liver, spleen, testes, and kidneys, leprosy bacilli were found when no naked-eye changes could be detected. This accords with Cornil and Babes' observations already quoted. Indeed, the writer has never seen any visceral macroscopic changes which could with certainty be ascribed to leprosy.

Meanwhile the general axiom holds good, that in every case of leprosy, during a part or the whole of its course, leprosy bacilli are to be found if a long enough search is made for them. Whether any or what viscera are invaded in a given case seems, however, to be subject to no fixed law.

Table containing a Summary of One Hundred and Nine Necropsies on Lepers.

No.	Birth-place.	Colour.*	Age. M	Age. F.	Date of death.	Form of leprosy.†	Duration in years.	Cause of death.	Distribution of bacilli.	Post-mortem appearances.
1	India	C	40	—	Apr. 10, 1884	A	7	Phthisis	—	Cavities in right middle lobe. Pneumonia of left base.
2	China	C	46	—	Apr. 13, 1884	A	—	Mixed kidney	—	Pleuræ adherent to right base. Interstitial and tubal nephritis. Spleen large and soft. Necrosis of nasal bones. Small abscess in left orbital lobule.
3	Trinidad	C	21	—	May 9, 1884	T	8	Large white kidney	—	Tuberculated ulcers on both legs. Kidneys 9 oz. each; large white. Spleen 26 oz.; hard. Cicatrix on inner surface. Patch of consolidation in upper lobe of right lung. Pleuræ adherent.
4	India	C	23	—	June 30, 1884	T	—	Granular kidney. Syphilis	—	Ulceration and commencing gangrene of left foot. Femoral glands suppurating. Left pleura adherent. Small gummata in kidneys, liver, and spleen. Right kidney 1¼ oz.; left 2¼ oz. Cortices almost gone. Testes small; fibrous tissue increased, and compressing tubules. Gummatous mass round seventh pair of nerves at exit from skull. Thickening of epiglottis with erosion of cartilage.
5	Trinidad	C	—	23	July 9, 1884	T	—	Cirrhosis of liver. Large white kidney	—	Liver 4¼ lbs., tough; commencing cirrhosis, and a good deal of fat. Spleen 19 oz.; simple hypertrophy. About a gallon of fluid in abdomen. Kidneys 10 oz. and 8 oz. Both pleuræ adherent.
6	Trinidad	C	—	21	Dec. 20, 1884	A	—	Ulceration	—	Legs and feet ulcerated and becoming gangrenous. Superficial ulceration of upper extremities and face. Left pleura slightly adherent. Kidneys fatty.
7	Trinidad	B	18	—	Dec. 22, 1884	T	—	Gangrene	—	Gangrene of both legs. Pleuræ slightly adherent at bases. Old adhesions between spleen and diaphragm. Fibro-calcareous nodule in spleen with depressed scar over it.

* W = White; B = Black; C = Coloured. † T = Tuberculated; A = Anæsthetic; M = Mixed.

No	Birth-place.	Colour.	Age.		Date of death.	Form of leprosy.	Duration in years.	Cause of death.	Distribution of bacilli.	Post-mortem appearances.
			M.	F.						
8	Africa	B	—	45	Dec. 26, 1884	A	1	Gangrene	—	Moist gangrene of both legs. Spontaneous separation of left foot. Dry gangrene of nose, fingers, knees, and right great toe. Liver and kidneys fatty.
9	Martinique	B	—	44	Jan. 3, 1885	M	10	Exhaustion	None in median nerve	Toes ulcerated. Median nerves thickened above wrists. A few ounces of turbid serum in left pleura. Old lymph in right lobe of liver.
10[1]	Trinidad	C	—	41	Feb. 9, 1885	M	—	Cirrhosis of liver	Numerous in larynx	Legs and hands ulcerated. Liver 36 oz.; cirrhosis. Spleen adherent to diaphragm. Some pints of fluid in abdomen. Superficial ulceration of colon. Mucous membrane of larynx thickened; ulcers over epiglottis and ventricles.
11[2]	Trinidad		23	—	Mar. 10, 1885	T	13	Lardaceous degeneration. Phthisis	Generally diffused in larynx. None at apex of left lung	Ulceration of hands, feet, and face. Liver 6½ lbs.; lardaceous. Kidneys 12 oz.; lardaceous. Spleen 18 oz.; hypertrophied. Small caseous mass at apex of left lung, with a zone of grey tubercles. Superficial ulceration of large intestine. Tubercles on tongue, anterior pillars of fauces, epiglottis, and arytænoids. Aryepiglottic folds swollen and ulcerated, and rima narrowed.
12	Trinidad	C	—	16	Mar. 15, 1885	T	—	Phthisis	Numerous in caseating femoral gland. None in lung	Ulceration of nose and ears. Skin of extremities separating in large patches. Left femoral glands enlarged, caseating. Cavity about 3 inches in diameter at apex of right lung; broncho-pneumonia at right base. Pleuræ thickened, adherent. Superficial ulceration of large intestine.
13	Trinidad	B	19	—	Mar. 16, 1885	T	7	Mixed kidney	None in femoral gland or kidney	Fingers swollen, ulcerated. Left pleura slightly adherent. Kidneys 12 oz.; cortices blurred; surface granular. Liver 51 oz.; fatty. Spleen 16 oz.; hypertrophied. About 3 pints of fluid in abdomen.
14	Trinidad	B	—	30	Mar. 16, 1885	T	7	Large white kidney	Generally diffused in femoral gland and tubercle of nose None in kidney	Femoral glands size of walnuts. Left pleura adherent. Large intestine congested and ulcerated. Kidneys 13 oz.; large white. Small cysts in ovaries.
15[3]	India	C	47	—	Mar. 23, 1885	M	13	Gangrene	None in femoral gland or median nerve	Extensive gangrene of left foot. Superficial ulceration of nose and ears. Fingers swollen and ulcerated. Femoral glands enlarged. Large fusiform swelling of left median nerve above wrist. Right less enlarged.

16	Trinidad	C	25	—	April 5, 1885	T	9	Large white kidney	Numerous in larynx and femoral gland. None in kidney or ulcer	Superficial ulceration of face, ears, and extremities. Femoral glands enlarged. Ragged ulceration of pharynx. Epiglottis thickened and ulcerated. Kidneys 13 oz.; large white, mottled and fatty. Large intestine reddened and superficially ulcerated.
17[4]	Trinidad	W	22	—	May 14, 1885	M	—	Phthisis	Numerous in larynx, femoral gland, and tubercle of ear. Few in lung	Second and third phalanges of fingers separated spontaneously. Femoral glands enlarged. Epiglottis and larynx thickened and ulcerated. Lungs contain numerous cavities with fœtid pus and blood-clots; lungs gangrenous in places. Spleen 16 oz.; hypertrophied.
18[5]	Trinidad	C	14	—	May 31, 1885	T	2	Pyæmia[7]	None in femoral gland or pulmonary infarct	Flaps united well; drainage-tube still in. About ¾ inch of femur bare and stripped of periosteum; around this a cavity filled with fœtid pus. Pus extends 2 inches up medulla of femur. About half a pint of purulent lymph in right pleura. Base of right lung contains irregular infarcts about the size of small plums. Femoral glands enlarged. Mucous membrane of bladder healthy; no evidence of lymphatic disease. No filariæ found. Circular ulcer about an inch deep over right buttock.
19	Trinidad	C	17	—	June 10, 1885	M	6	Phthisis	Numerous in lung. Few in tubercle of ear and femoral gland. None in median nerve	Face and ears tuberculated. Old separation of fingers and toes. Left lung completely excavated; blood-vessels traversing cavity, covered with thick purulent deposit. Right lung has cavity at apex, size of orange; rest of lung thickly studded with miliary tubercles to extreme base, caseating in places.
20[6]	India	C	35	—	June 11, 1885	A	—	Acute abscess. Hæmorrhage after amputation of arm	None in median nerve or spinal cord	Pus extending along axillary vessels into posterior triangle, which was converted into an abscess cavity, the nerves of the cervical plexus being dissected out by the suppuration. The abscess had also travelled down along the scalenus anticus, and was pointing at the apex of the right lung. Pleura slightly adherent here. Spleen adherent; old scar on outer surface.
21	India	C	47	—	June 13, 1885	A	23	Large white kidney	None in femoral gland, spleen, liver, or kidney	Bullæ and punched-out ulcers on lower extremities. Kidneys 11 oz.; large white. Spleen 9 oz.; hypertrophied. Much fluid in left pleura, with recent lymph towards base. Some pints of fluid in abdomen.

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[1] 'Path. Soc. Trans.,' vol. xxxvii. [2] Ibid. [3] Ibid. [4] Ibid. [5] 'Lancet,' 1890. [6] 'Brit. Med. Journ.,' 1885.

[7] Following amputation through thigh for elephantiasis Arabum, from which patient suffered in addition to leprosy.

No.	Birth-place.	Colour.	Age.		Date of death.	Form of leprosy.	Duration in years.	Cause of death.	Distribution of bacilli.	Post-mortem appearances.
			M.	F.						
22[1]	Trinidad	B	—	19	June 14, 1885	A	6	Phthisis	Few in lung and femoral gland	Old contraction of fingers and separation of phalanges. Left lung completely excavated. Right lung filled with grey and yellow caseating tubercles from apex to base.
23[2]	Grenada	B	14	—	June 18, 1885	A	1	Phthisis	Generally diffused in larynx. Few in femoral gland. None in lung, brain, or mediastinal gland	Ragged ulceration of larynx. Vocal cords completely gone. Thyroid cartilages separated and laid bare; perforating ulcer leading between them to an abscess cavity outside. Cavities size of an orange at right apex and base, also at left apex. Lungs full of tubercles. Caseous and calcareous glands in anterior mediastinum.
24	Trinidad	B	70	—	June 28, 1885	M	4	Gangrene	Numerous in femoral gland, testes, and larynx. Few in tuberculated feet. None in median nerve or liver	Hands gangrenous. Large ulcer on right leg. Toes greatly hypertrophied and deformed. Desquamation all over body. Median and ulnar nerves thickened above wrists. Ulnar nerves enlarged behind internal condyles. Mucous membrane of epiglottis and larynx thickened. Femoral glands about 2 inches long.
25	Trinidad	W	—	50	June 30, 1885	A	10	Exhaustion	Few in median nerve. None in liver	Body extremely wasted. Old separation of phalanges of fingers. Slight thickening of median nerves. Some adhesions of pleuræ. Aorta atheromatous.
26	Trinidad	B	16	—	July 15, 1885	A	7	Phthisis	None in femoral gland, lung, or median nerve	Old separation of phalanges of right hand. Slight thickening of median nerves. Femoral glands about 1½ inches long. Cavity at apex of right lung about 6 × 3 inches; another at base about 3 inches diameter. Cavity at apex of left lung about 3 × 4 inches. Rest of lungs full of miliary tubercles. Superficial ulceration of larynx and epiglottis.
27	India	C	—	65	July 26, 1885	A	1	Dysentery	Generally diffused in femoral gland. None in kidney, spleen, liver, or median nerve	Fingers and toes ulcerated. Fracture of right clavicle; no callus. Slight pleuritic adhesions. Congestion and superficial ulceration of large intestine. Femoral glands enlarged. Some thickening of median nerves.
28	Trinidad		19	—	July 28, 1885	T	8	Gangrene	Few in spleen and liver. None in median nerve, femoral gland, or	Sloughing ulcer involving lower half of left leg; about 6 inches of fibula bare and white. Sloughing ulcer on dorsum of left foot. Clean ulcer on front of right ankle. Fingers ulcerated. Femoral glands much enlarged. Thick-

									kidney	ening and superficial ulceration of mucous membrane of larynx. Spleen 13 oz.; hypertrophied. Some adhesion of left pleura.
29	Trinidad	B	25	—	Aug. 18, 1885	A	—	Facial carbuncle. Pyæmia	None in kidney, liver, spleen, pons, median nerve, or infarct of lung	Brawny infiltration of left cheek extending behind ear. Burrowing of pus in subcutaneous tissues. Eyeballs prominent; thrombosis of both ophthalmic veins. Cavernous sinuses filled with clots and sanious pus. Thrombosis extends to lateral and superior petrosal sinuses. Yellow lymph on pons and cerebellum. Pyæmic infarcts about size of filberts breaking down at bases of lungs. A little yellow lymph on pleura. Spleen 17 oz.; some lymph on surface.
30	India	C	33	—	Aug. 22, 1885	A	—	Mottled kidney. Gangrene of lung	None in lung, spleen, kidney, liver, testes, or femoral gland	Commencing gangrene of left foot and wrist. Right pleura adherent. Left lung œdematous; hæmorrhage into tissue. Cavities size of filberts, filled with fœtid pus; commencing gangrene. Kidneys 14 oz.; fatty; hæmorrhages beneath mucous membrane of pelvis and in cortices. Spleen 16 oz.; slight thickening of capsule. Femoral gland about 2 inches long.
31	Trinidad	C	15	—	Aug. 28, 1885	T	—	Large white kidney	Few in thickened lobe of ear. None in liver, spleen, kidney, ulnar nerve, or femoral gland. Few doubtful in testes	Ulceration of face. Commencing gangrene of fingers, legs, and feet. Liver very fatty. Kidneys 13 oz.; surface smooth, with patches of congestion. Microscope shows tubal nephritis. Large intestine very congested, especially over rugæ. Mucous membrane separates easily. Spleen 13 oz. Femoral glands about 2 inches long.
32[4]	Trinidad	C	60	—	Oct. 11, 1885	T	4	Syphilis. Cystitis. Suppression of urine	Generally diffused in tubercle of face. Numerous in femoral gland. None in liver, spleen, kidney, testes, or median nerve	Ulceration of posterior nares and back of pharynx. Tip of epiglottis ulcerated away. Scars on larynx and vocal cords. Small patch of recent pneumonia at right apex. Bladder thickened; mucous membrane greenish black, coated with phosphatic deposit, especially at orifices of ureters. Bladder contains a few drachms of grumous ammoniacal urine. Straw 4¼ inches long at neck of bladder, coated with phosphates. Testes fibrous; right epididymis replaced almost entirely by cartilage; left also cartilaginous and becoming calcified. Kidneys 10 oz.; normal. Nose sunken from old disease of bones.

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[1] 'Path. Soc. Trans.,' vol. xxxvii.

[2] Ibid.

[3] 'Brit. Med. Journ.,' 1886.

[4] 'Path. Soc. Trans.,' vol. xxxvii.

No.	Birth-place.	Colour.	Age.		Date of death.	Form of leprosy.	Duration in years.	Cause of death.	Distribution of bacilli.	Post-mortem appearances.
			M.	F.						
33	India	C	48	—	Nov. 13, 1885	M	9	Atheroma of heart. Cirrhosis of liver	Numerous in tubercle of nose. None in lobe of ear, median nerve, femoral gland, liver, spleen, kidney, or lung	Right hand and lower part of forearm œdematous. Ulceration of fingers, toes, and nose. Heart 10 oz.; sharp calcareous masses at all four valvular orifices, especially above mitral and aortic valves. Valves and heart substance healthy. Liver 47 oz.; very tough, nodular on surface. Femoral glands about 2 inches long.
34[1]	Trinidad	B	28	—	Nov. 27, 1885	A	4	Phthisis	None in spleen, liver, kidney, brain, median nerve, femoral gland, bronchial gland, lung, or pus from abscess	Old contraction of fingers. Both lungs full of miliary tubercles. At left apex an irregular cavity about the size of an orange, containing blood-clots. Walls studded with grey tubercles. Smaller cavities filled with greenish pus. Heart 12 oz.; hypertrophied. Kidneys 10 oz.; healthy. Left femoral gland about 3 inches long.
35	Trinidad	C	—	9	Nov. 29, 1885	T	—	Lardaceous degeneration	None in spleen, liver, kidney, femoral gland, median nerve, or tubercle of ear	Superficial excoriation of trunk and extremities. Spleen 1 oz.; hard, lardaceous. Kidneys 3 oz.; less lardaceous. Liver 3 lbs.; very hard, lardaceous. About a pint of fluid in abdomen.
36	Trinidad	B	—	64	Feb. 5, 1886	A	20	Pyæmia. Pneumonia	None in femoral gland, liver, spleen, kidney, median nerve, or discharge from ulcer	Commencing gangrene in stump of left leg. No pus in medulla of bone or femoral vein. Femoral glands not enlarged. A few ounces of purulent lymph in left pleura. Dark solidified infarct with pale margin at extreme base of left lung, about 2 × 1 inches. Another about $\frac{3}{4}$ inch diameter a little higher up. Other viscera healthy. Slight enlargement of median nerves. Old separation of phalanges of fingers and toes.
37[2]	Trinidad	B	17	—	Feb. 8, 1886	T	7	Acute nephritis.	Numerous in tubercle of face.	Left kidney and adrenal together $6\frac{2}{3}$ drachms. Kidney alone estimated at 4 drachms. Right kidney and adrenal 8 oz.

								Œdema of lung	None in femoral, mesenteric, or renal glands, or in kidney, spleen, liver, testes, brain, or median nerve	Minute extravasations beneath mucous membrane of pelvis. Extravasations about ½-inch long beneath anterior surface of capsule. Microscopic examination showed the tubules stuffed with blood-corpuscles, granular cells, and débris. The outline of the epithelium could not be made out. The glomeruli were filled with blood extravasation and hyaline fibrinous material. These changes were less marked in the left kidney. Left ureter pervious. Bladder healthy. Heart 9 oz.; left ventricle firmly contracted; walls four times diameter of those of right. Lungs dark purple, sink in water, easily lacerable with finger. Œdema extreme at bases. Spleen 19 oz.; simple hypertrophy. Other viscera healthy.
38	Demerara	W	34	—	Feb. 17, 1886	M	20	Pleuro-pneumonia	Numerous in larynx, many in cells. Numerous in femoral gland. Generally diffused in testes. None in pulmonary abscess	Superficial ulceration of hands, nose, and neck. Granulating surface over stump of thigh, about 3 × 2 inches. Longitudinal section of femur shows compact tissue normal at condyles, but higher up it is thinned and encroached on by dark red diffluent medulla. Femoral glands about 1½ inches long. Tip of epiglottis ulcerated away. Vocal cords replaced by a ragged mass of granulations. Ulceration extends also above and below cords. Base of right lung completely solid, with the exception of a few small abscesses. Higher up a separate abscess about 1 inch diameter, with very thick walls. Yellow lymph and several ounces of bloody fluid in right pleura. Spleen 30 oz.; simple hypertrophy. Other viscera normal.
39	Trinidad	C	—	25	Mar. 6, 1886	A	15	Phthisis	None in liver, kidney, spleen, median nerve, tubercle of lung, or medulla of tibia	Old separation of phalanges of all fingers, and thumbs of both hands. In left foot only heel remains, with sinus leading to carious bone. Longitudinal section shows ankylosis of remains of calcaneum to tibia. Cancellous tissue of tibia almost replaced by medulla. Compact tissue much thinned. In right foot old spontaneous separation of all toes except great. Upper lobes of lungs filled with caseating tubercles. Median nerves not enlarged. Other viscera healthy.

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[1] 'Brit. Med. Journ.,' 1885.

[2] 'Path. Soc. Trans.,' vol. xxxvii.

[3] 'Brit. Med. Journ.,' 1885 and 1890.

No.	Birth-place.	Colour.	Age.		Date of death.	Form of leprosy.	Duration in years.	Cause of death.	Distribution of bacilli.	Post-mortem appearances.
			M	F.						
40	Trinidad	C	23	—	Mar. 18, 1886	M	12	Mixed kidney. Œdema of lungs	Generally diffused in larynx. Few in femoral gland. None in liver, kidney, spleen, or median nerve	Median nerves thickened above wrists. Ulceration of legs and commencing gangrene of fingers, fauces, and larynx, Tip of epiglottis ulcerated away. Numerous small ulcers on larynx and vocal cords. Recent lymph and several ounces of serum in each pleura. Lungs œdematous. Kidneys 12 oz.; surface scarred and irregular. Cortices blurred; fatty in some places, with patches of congested capillaries in others. Stomach dark, with capillary ecchymoses. Other viscera healthy.
41	India	C	21	—	April 3, 1886	A	6	Abscess of liver. Pleurisy	Generally diffused in median nerve. None in liver, kidney, spleen, or femoral gland	Legs œdematous. Perforating ulcer of right sole. Commencing gangrene of left hand. Right median nerve enlarged. Pleuræ adherent; considerable fluid in each. Recent lymph at right base, with pus in tubes. Liver 29 oz.; near portal fissure is an abscess about $\frac{3}{4}$ inch in diameter, filled with thick pus. Near it are smaller abscesses, and a fibrous cicatrix with caseous material. No pylephlebitis or ulceration of stomach or intestines. Spleen 9 oz.; hard; no stain with iodine.
42	Trinidad	B	23	—	May 6, 1886	T	5	Phthisis. Pneumothorax	Numerous in femoral gland. None in liver, kidney, spleen, lung, or median nerve	Gangrene of left foot. Superficial ulceration of hands and buttocks. Right median nerve thickened above wrist. Upper half of right lung excavated. Base forced up from diaphragm by large cavity with recent lymph on walls, and containing some fluid. The perforation could not be found. Numerous yellow tubercles in left lung, in some places breaking down into cavities size of filberts. Other viscera healthy. Femoral glands enlarged.
43	India	C	37	—	Oct. 31, 1886	M	13	Phthisis	None in liver, spleen, kidney, testis, tubercle of lung, median nerve, or enlarged femoral gland	Gangrenous ulceration of left foot. Ulceration of hands and over trochanters. Lungs full of small grey tubercles. Left breaking down at apex. Pleuræ very adherent. Spleen 14 oz.; hypertrophied; capsule thickened. Small intestine deeply congested. Solitary follicles of large intestine swollen, and surrounded by rings of deep purple injection. Left femoral glands caseous, breaking down in centre. Other viscera healthy.

44[1]	Germany	W	—	60	Nov. 14, 1886	A	12	Pneumonia	None in kidney, spleen, liver, sciatic nerve, or femoral gland. A few doubtful in lung	Sacculated aneurism of about 3 inches in diameter, springing from ascending and transverse aorta. Slightly flattened against manubrium, which it had eroded to the depth of about ¼ inch. Filled with regular layers of distinctly laminated clot, breaking down into grumous material towards the centre. Opening from sac into aorta filled by two rounded bosses of fibrin. Aorta covered with small patches of atheroma. Advanced grey hepatisation of left base; firm adhesions of right pleura. Abscess over right wrist and swelling and redness of right leg. Fingers shortened and incurved. Ulcer in left foot. Other viscera healthy.
45	India	C	46	—	Nov. 17, 1886	M	9	Emphysema. Gangrene	None in kidney, liver, lung, median nerve, femoral gland, or ante-mortem clot. Numerous large and small cells in spleen full of bacilli. Few similar ones in sciatic nerve	Gangrenous ulcers on backs of hands and over right knee. Œdema of left upper extremity to shoulder. Lungs generally emphysematous. Heart 8 oz.; firm patch of partly decolourised clot in left ventricle. Spleen 13 oz.; simple hypertrophy. Other viscera healthy.
46	Trinidad	B	—	15	Jan. 19, 1887	T	4	Phthisis	Numerous in tubercle of ear. Generally diffused in intestinal ulcer, larynx, femoral gland, tubercle of lung, and cells of spleen and liver	Ulcerated tubercles on face and ears. Small ulcers on back of epiglottis, over arytænoids, and in angle of thyroid. Mucous membrane of larynx swollen. Right lung full of tubercles; upper lobe becoming excavated. Left lung hollowed out into one large sac, containing much pus. Numerous punctiform ulcers in small intestine.
47	Trinidad	B	—	18	Jan. 19, 1887	T	5	Large white kidney. Tuberculosis	Numerous in femoral gland. None in median nerve, kidney, liver, or tubercle of pleura	Ulcerated tubercles of face and ears. Tubercles on pleura over right base, also between lobes of lung. Liver 67 oz.; lardaceous. Kidneys 24 oz.; large white. Right median nerve thickened. Femoral glands enlarged.

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[1] 'Path. Soc. Trans.,' vol. xxxviii.

No.	Birth-place.	Colour.	Age.		Date of death.	Form of leprosy.	Duration in years.	Cause of death.	Distribution of bacilli.	Post-mortem appearances.
			M.	F.						
48	Trinidad	W	48	—	Jan. 30, 1887	A	34	Mixed kidney	None in liver, spleen, kidney, testis, femoral gland, or sciatic nerve	Sores on buttocks. Ulnar and median nerves atrophied. Spleen 17 oz.; capsule thickened; yellow and white masses and scars. Right kidney 3 oz.; left 6 oz.; deeply scarred. Capsule quite gone in places; in others evident tubal change.
49	Africa	B	54	—	Feb. 8, 1887	A	25	Cirrhosis of liver. Contracted kidney. Tubercle of lungs and peritoneum	None in anterior dorsal and cervical roots, kidney, liver, tubercle of mesentery, or of lung	Much cerebral effusion. Ulnar nerves thickened at elbows. About a pint of fluid in left pleura. Lungs full of miliary tubercles. Heart 11 oz.; right ventricle hypertrophied. Liver 24 oz.; hob-nailed, very tough; mottled with yellow patches on section. Kidneys 7 oz.; capsules adherent; cortices deeply scarred. Mesentery and peritoneal surface of intestines covered with small translucent nodules. About 5 pints of fluid in abdomen.
50	Africa	B	—	70	Feb. 16, 1887	A	13	Cancer of uterus	None in kidney, liver, spleen, femoral gland, growth in uterus, or median nerve	Median and ulnar nerves atrophied. Ulceration of feet and hands and old separation of fingers. Pleuræ very adherent. Aorta atheromatous. Liver adherent to diaphragm. Uterus enlarged; cavity ragged, ulcerated. Walls thickened, and containing irregular masses and bosses of new growth, showing large round and pyriform cells under microscope. An opening had ulcerated from the uterus into the ileum, and the uterus contained fæcal matter. A gaping blood-vessel was found in the wall of the uterus filled with a firm cylinder of clot. Vagina gaping; ulcerated about orifice. No secondary deposits. Femoral glands enlarged; ordinary lymphoid cells found.
51	India	C	42	—	Feb. 17, 1887	M	8	Obstruction of larynx	Numerous in larynx. Generally diffused in femoral gland. Few in spleen. None in liver, kidney, median nerve, sciatic nerve, or testis	Superficial ulceration of feet and stumps of fingers. Bedsore over right buttock. Epiglottis thickened, tuberculated, ulcerated at apex. Ulcerating tubercles over arytænoids and ary-epiglottic folds. Cords much ulcerated, and considerable ulceration and commencing gangrene of mucous membrane below cords. Bases of lungs extremely congested. A few ounces of fluid in pleuræ. Median nerves much thickened, and of a dull grey colour above wrists.

52	Trinidad	B	31	—	Mar. 2, 1887	T	9	Phthisis	Numerous in cells of spleen, liver, larynx, and femoral gland. Numerous singly in testes. None in kidney, phthisical lung, or median nerve	Median nerves much swollen above wrists, especially left; semi-translucent and greasy-looking. Mucous membrane of larynx much thickened; ulceration over vocal cords and tip of epiglottis. Left lung consolidated at apex; contains several cavities in which are recent blood-clots. Numerous tubercles lower down. Left pleura very adherent. Right lung contains a few scattered grey tubercles. Nipples tuberculated.
53	Nevis	B	27	—	Mar. 4, 1887	M	5	Phthisis. Pneumothorax	Few in phthisical lung. Mesenteric and femoral glands. None in spleen, kidney, testes, median nerve, or caseous nodule of liver	On opening right pleura air rushed out audibly, and lung was found compressed towards spine. About a pint of dirty fluid in pleura. Large irregular cavity involving greater part of upper lobe of right lung, containing much dirty purulent material, and towards axilla communicating with pleural cavity by a small round opening rather larger than a pin's head. Pleura not adherent. Patches of consolidation and numerous tubercles around cavity. Lower lobe almost free from tubercles. Cavity at left apex about the size of a walnut. Tubercles starting from it. Lower lobe healthy. Liver contains numerous caseous tubercles from ¼ to ½ inch in diameter, especially towards upper and lower surfaces. Kidneys 4 oz each; capsules adherent; cortices blurred, deeply scarred. Large mass of caseous mesenteric glands near spine, breaking down into pus. Left median nerve thickened.
54	India	C	36	—	Mar. 17, 1887	A	11	Tuberculosis of lungs. Chronic pleurisy	None in liver, kidney, spleen, testes, median nerve, lung, or femoral gland	Remains of yaw tubercles on face and extremities. Encysted pleurisy at left base, with septa running across, and about a pint of reddish fluid. Pleura fleshy; much thickened. Lung pressed up; full of small grey tubercles. Right lung contains numerous scattered grey tubercles.
55	Trinidad	B	27	—	April 5, 1887	A	17	Mixed kidney. Lardaceous liver	None in liver, spleen, median nerve, testis. Doubtful in lung	Old separation of fingers and toes. Much effusion in right ankle and tarsal joints. Several pints of fluid in abdomen. Left pleura very adherent in front; a quantity of fluid behind. Two or three patches of broncho-pneumonia. Spleen 11 oz.; hypertrophied. Liver 6 lbs.; lardaceous. Kidneys 10 oz.; capsules adherent; cortices thinned, blurred, mottled with yellow.

No.	Birth-place.	Colour.	Age.		Date of death.	Form of leprosy.	Duration in years.	Cause of death.	Distribution of bacilli.	Post-mortem appearances.
			M.	F.						
56[1]	Trinidad	B	13	—	April 6, 1887	T	3	Pyæmia	Few in liver, spleen, and mesenteric gland	Some pints of fluid in pleura and peritoneum. Liver 30 oz. Spleen 10 oz. Kidneys 6 oz. These three viscera contain numerous small infarcts, varying in size from a pin's head to a small shot. Five sloughing ulcers in cæcum; edges undermined and muscular coat exposed. Mesenteric glands swollen, translucent, containing small yellow points.
57	India	C	—	52	April 13, 1887	M	5	Tuberculosis. Large white kidney	Generally diffused in larynx and femoral gland. None in liver, spleen, kidney, median nerve	Median nerves thickened. Femoral glands much enlarged. Epiglottis and larynx congested and ulcerated. Lungs full of miliary tubercles. Tubercles on pleuræ. Kidneys 16 oz.; large white; contain small yellow tubercles. Spleen 16 oz.; contains yellow masses about ¼ inch in diameter. Generally diffused tubercles in liver, omentum, and peritoneum. Much fluid in latter.
58	Trinidad	B	17	—	April 20, 1887	T	5	Pyæmia. Tuberculosis of lungs	Generally diffused in femoral gland and epiglottis. None in liver, testis, lung, or sciatic nerve	Body wasted. Much absorption of tubercles. Gangrenous ulcer 5 inches long at back of right leg. Median nerves much thickened at wrists. Epiglottis thickened. Lungs full of miliary tubercles. Heart 9 oz.; hypertrophied; small yellow patches on aorta above valves. Spleen 16 oz.; three large infarcts about 1 inch in diameter under capsule. Kidneys 11 oz.; infarcts about ⅓ inch long in cortices. Few small yellow nodules on liver near portal fissure. Femoral glands enlarged.
59	Trinidad	C	22	—	May 6, 1887	M	7	Gangrene	Few in liver and spleen, chiefly in cells. None in infarct of lung, femoral gland, median nerve, or kidney	Gangrenous ulcers involving greater part of both feet. Fingers ulcerated. Median nerves thickened above wrists. Abscess in right lung about size of cob-nut; walls thickened. Small patch of consolidation higher up. Femoral glands enlarged.
60[2]	France	W	49	—	May 19, 1887	A	5	Abscess of liver. Dysentery	Numerous free and in cells in median nerve and superior cervical	Median nerves thickened above wrists. A few ounces of fluid in each pleura. Aorta very atheromatous. Liver 83 oz.; has five distinct abscesses, containing altogether from 2 to 3 pints of pus. Circular sharp-cut ulcers throughout large

									ganglion of sympathetic. Few in liver. None in spleen, kidney, femoral gland, or ganglion of vagus	intestine. Kidneys 9 oz.; cortices thinned, blurred, granular on surface. Spleen 18 oz.; hypertrophied.
61	Trinidad	B	—	12	May 28, 1887	T	3	Large white kidney. Pyæmia	Numerous in femoral gland. Few in ulnar nerve at elbow. None in infarct of spleen, liver, kidney, or lung	Œdema, redness and thickening of tissues above ankles. Median and ulnar nerves thickened at wrist and elbow respectively. Chain of enlarged, caseous, suppurating glands behind each sterno-mastoid. Pus burrowing in each posterior triangle. Right pleura full of fluid; lung tough, compressed. A little fluid in left pleura. Several infarcts in spleen and kidneys, from ¼ to ¾ inch in diameter. Liver 28 oz.; tough, surface irregular; a few globular yellow masses, the size of small shot. Femoral glands enlarged. About 2 pints of fluid in abdomen.
62	Trinidad	B	19	—	June 10, 1887	A	15	Large white kidney	Few in thickened median. None in spinal cord, femoral gland, infarct, or abscess of lung, liver, spleen, testis, or abscess of intestine	Enormous enlargement of right median nerve, commencing about upper third and gradually increasing down to wrist. Nerve gelatinous-looking, and fibres greatly swollen and brown. Right pleura very adherent. Old abscess in upper part of right lung, about ½ inch in diameter; walls thickened. Numerous recent dark red infarcts in left lung ¼ to ½ inch in diameter. Kidneys 16 oz.; large white, with some lardaceous degeneration. Numerous small abscesses scattered along small intestine; fewer in large. Femoral glands enlarged.
63	China	C	66	—	June 20, 1887	A	2	Gangrene	Few in median nerve. None in liver, kidney, spleen, lung, femoral gland, or testis	Gangrene of right foot, extending from ulcer. Lower extremity almost black as high as Poupart's ligament. No thrombosis in femoral vein. Median nerves thickened above wrists. Slight adhesions of pleura; hard fibrous nodule near upper part of right lung. In left kidney a cyst about the size of a walnut, containing cholesterin, and smooth, hard projections from walls. Microscope shows dense fibrous tissue. Other smaller cysts in both kidneys containing smaller masses. Femoral glands enlarged.

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[1] 'Brit. Med. Journ.,' 1887.

[2] 'Lancet,' 1887.

No.	Birth-place.	Colour.	Age.		Date of death.	Form of leprosy.	Duration in years.	Cause of death.	Distribution of bacilli.	Post-mortem appearances.
			M.	F.						
64	India	C	73	—	July 2, 1887	M	5	Obstruction of larynx	Cells full of bacilli in larynx, femoral gland, liver, and spleen. None in median nerve, testis, or kidney	Median nerves thickened above wrists. Superficial ulceration of epiglottis, larynx, and vocal cords. Lungs dark purple at bases; much fluid escapes on section. Liver 46 oz.; lardaceous. Kidneys 4 oz.; cortices thin and surface irregular. Encysted hydrocele in each testis, containing much cholesterin. Much cartilage round left hydrocele. Tubular part of testis diminished.
65	Germany	W	60	—	July 5, 1887	M	11	Gangrene of lungs	Generally diffused in liver and kidney. None in femoral gland or median nerve	Median nerves thickened. Ends of fingers separating. Lungs diffluent, collapsing when cut into. Fœtid, dirty fluid in large cavities of lung and in pleura. Large plate of cartilage 4 × 4 inches and $\frac{1}{4}$ inch deep on outer surface of spleen. Femoral glands enlarged.
66[1]	Africa	B	43	—	July 26, 1887	A	13	Strangulated hernia	Generally diffused in median nerve. None in liver, spleen, kidney, testis, or femoral gland	Lower coils of small intestine congested, and all large intestine dark purple. Few flakes of lymph on peritoneum. Some coils of small intestine were pulled out of the left tunica vaginalis without any difficulty. No ulceration and no adhesion. Inguinal canal greatly dilated. On slitting up the small intestine it was found to be congested for about the extent of the ileum. The large intestine was intensely congested, the mucous membrane being ulcerated, and large clots of blood standing out on its surface. No perforations. Median nerves somewhat thickened.
67	Trinidad	B	21	—	July 27, 1887	M	17	Obstruction of larynx	Numerous in larynx and femoral gland. Generally diffused in median nerve. Few in tongue, testis, and thickened skin from leg. In liver one	Nose sunken from disease of bones. Median nerves thickened. Femoral glands enormously enlarged. Tongue thickened. Epiglottis almost destroyed by old ulceration. The upper orifice of the larynx is narrowed by cicatricial contraction to a circle about $\frac{1}{2}$ inch in diameter. The remains of the epiglottis and arytænoids are dragged to one level. Vocal cords thickened and superficially ulcerated; general thickening of tissues of larynx. Small encysted hydrocele in each testis. Great increase of fibrous tissue in testis and

									cell full of bacilli. Numerous similar cells in spleen. None in kidney	epididymis.
68	India	C	46	—	Aug. 10, 1887	A	1	Mixed kidney	None in liver, spleen, kidney, testis, femoral gland, or median nerve	Œdema of extremities. A few adhesions of right pleura. Kidneys 7 oz.; cortices thinned and scarred. Surfaces covered with small cysts. Capsules adherent. Liver 29 oz.; surface irregular; tissue deeply bile-stained; commencing contraction. Mucous membrane of intestines sodden; no bile. Large intestine loaded with mucus.
69	India	C	53	—	Sept. 17, 1887	A	—	Hip disease	Generally diffused in liver, spleen, and femoral gland. None in kidney, pons, or median nerve	Capsule of hip ruptured and ligamentum teres destroyed. Acetabulum and head of femur extensively eroded. Cartilage exfoliating. Muscles round joint matted together by inflammatory effusion and burrowed by pus. A few old adhesions of left pleura.
70	Trinidad		34	—	Oct. 24, 1887	M	15	Obstruction of larynx	Numerous in larynx and median nerve. Generally diffused in liver. Few in spleen, kidney, and femoral gland	Mammæ much developed. Skin of extremities very hard, thick, wrinkled, and scaly. Median nerves thickened. Tongue much enlarged. Great thickening of mucous membrane over epiglottis, ary-epiglottic folds, and vocal cords. Lungs congested; lower lobe on each side partly consolidated. Liver 94 oz.; lardaceous. Spleen 15 oz.; hypertrophied and adherent to diaphragm. Femoral and inguinal glands much enlarged. Small encysted hydroceles in testis.
71[2]	India		40	—	Nov. 16, 1887	T	3	Ankylostomiasis. Tuberculosis	Numerous in median nerve and femoral gland. Few in testis. None in tubercle of lung, liver, or spleen, nor in kidney, brain, or intestinal mucus	Median nerves thickened. Separate fasciculi enlarged in places. Scattered miliary tubercles in both lungs. Right pleura full of fluid; lung compressed. Some fluid in left pleura. Liver 31 oz.; contains numerous miliary tubercles. Spleen 6 oz.; contains similar tubercles. Numerous ankylostomata and small fresh hæmorrhages in upper half of jejunum. Much dark blood in ileum. Considerable fluid in abdomen.

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[1] 'St. Louis Med. and Surg. Journ.,' 1888.

[2] 'Path. Soc. Trans.,' vol. xxxix.

No.	Birth-place.	Colour.	Age.		Date of death.	Form of leprosy.	Duration in years.	Cause of death.	Distribution of bacilli.	Post-mortem appearances.
			M.	F.						
72	Trinidad	B	16	—	Dec. 8, 1887	T	8	Large white kidney	Generally diffused in liver and spleen. Few in femoral gland. None in kidney, testis, or median nerve	Symmetrical circular bedsores, 2 inches in diameter, behind trochanters. Tissues over sacrum infiltrated. Median nerves thickened; fibres translucent and yellowish. Bases of lungs solid, with hæmorrhages the size of small cherries. Heart 5 oz.; left ventricle about 4 times thickness of right. Kidneys 9 oz.; large white; capsules strip easily. Liver 37 oz.; pale and very tough. Testes undeveloped; size of beans. Femoral glands enlarged.
73	Trinidad	B	36	—	Dec. 27, 1887	T	10	Obstruction of larynx. Ankylostomiasis	Numerous in larynx. Few in femoral gland and solitary follicle. None in liver, kidney, spleen, testis, or median nerve	Ulnar nerves at elbows and median nerves above wrists coarse and thickened. Epiglottis thickened; apex dragged down; patch of black pigment on anterior surface. Ragged ulceration of vocal cords. Both pleuræ adherent. Lungs filled with frothy fluid. Kidneys 8 oz.; very pale and sodden-looking. Spleen 26 oz.; numerous infarcts of various sizes. Numerous ankylostomata in duodenum, jejunum, and cæcum. Much blood and mucus in bowel. Solitary follicles swollen and dark purple, especially in lower part of jejunum. Femoral and inguinal glands enlarged.
74	Trinidad	B	51	—	Feb. 3, 1888	A	14	Mixed kidney. Tuberculosis of lungs	None in lung, liver, spleen, kidney, or femoral gland	Old separation of fingers and toes. Gangrene of right foot. Apices of lungs full of grey tubercles. Pleuræ adherent. Kidneys 10 oz.; pale white, with adherent capsules. Spleen 7 oz.; simple hypertrophy. Femoral glands enlarged.
75	India	C	57	—	Mar. 25, 1888	M	19	Granular kidney. Tubercular peritonitis	Generally diffused in femoral gland. None in liver, spleen, median nerve, or tubercle of omentum	Most of tubercles absorbed. Median nerves thickened. Abscess about ½ inch in diameter on lower surface of liver. Spleen 26 oz.; hypertrophied. Kidneys 5 oz.; capsules adherent; cortices thinned; surface extremely coarse and granular. Great omentum much thickened, and full of miliary tubercles. Parietal and visceral peritoneum covered with similar tubercles. Intestines adherent in places; mucous membrane sodden. About a pint of dark red dirty fluid in abdomen. Femoral glands enlarged.

76	Africa	B	46	—	Nov. 2, 1888	A	18	Phthisis	—	Old separation of fingers and toes. Perforating ulcer of left sole. Lungs full of tubercles. Cavities at right and left apices, size of orange and walnut respectively. Numerous smaller cavities filled with pus. Spleen 6 oz.; full of miliary tubercles. Median nerves slightly thickened. Left sciatic nerve shows no trace of former stretching.
77	Trinidad	C	45	—	Nov. 23, 1888	T	5	Suppuration. Hæmorrhage	Few in median nerve. None in liver, spleen, kidney, testis, or femoral gland	Tubercles still prominent on face. Stump of left arm burrowed by abscesses. Much breaking down blood-clot. Humerus bare. Right median nerve enlarged. Right pleura adherent. Spleen 11 oz.; hypertrophied. Several ankylostomata in duodenum.
78	Trinidad	B	—	18	Nov. 25, 1888	T	8	Sacro-iliac disease. Pyæmia. Granular kidney.	Numerous in caseous femoral gland. Few in caseous lumbar gland. None in spleen, liver, kidney, or median nerve	Body wasted. Nearly all tubercles absorbed. About ½ pint of fluid in left pleura. Kidneys 7 oz.; granular; capsules adherent. Spleen 5 oz.; numerous pyæmic infarcts, varying in size from a pin's heads to a small shot. Sinuses opening in lumbar region, when traced back over crests of ilia, are found to lead to an abscess in front of the sacrum, containing several drachms of pus. Tissues in front of vertebræ matted together with inflammatory material. Sacrum bare. Femoral and lumbar glands caseous.
79	Madeira	W	53	—	Jan. 23, 1889	M	7	Ulceration. Ankylostomiasis	Numerous in spleen and testis. Few in liver, kidney, and larynx. None in femoral gland and median nerve	Trunk and extremities covered with ulcers. Gangrene of fingers commencing. Tip of epiglottis ulcerated away. Ulceration of vocal cords and general thickening of mucous membrane. Small patch of consolidation at base of right lung. A little fluid in pleura. Aorta atheromatous. Liver fatty. Spleen enlarged. Seven ankylostomata in small intestine. Femoral glands enlarged.
80	Barbados	—	55	—	Feb. 16, 1889	A	1	Mixed kidney. Pleuritic effusion. Acute pericarditis	None in kidney, liver, spleen, femoral gland, testis, or median nerve	Body wasted. Œdema of extremities. Commencing contraction of fingers. Median nerves somewhat thickened. About two pints of fluid in left pleura; lung pressed against spine. Right pleura very adherent. Heart 15 oz.; much enlarged. Several ounces of fluid in pericardium; flakes of lymph on walls of heart. Spleen 9 oz.; simple hypertrophy. Cartilaginous nodule on surface. Kidneys 12 oz.; surfaces irregular; cortices thinned and blurred; numerous cysts, varying in size from small shot to hens' eggs. Aorta and right renal artery atheromatous.

No.	Birth-place.	Colour.	Age.		Date of death.	Form of leprosy.	Duration in years.	Cause of death.	Distribution of bacilli.	Post-mortem appearances.
			M.	F.						
81[1]	Trinidad	B	16	—	Feb. 19, 1889	T	5	Sunstroke	None in pons, median nerve, liver, spleen, femoral gland, or kidney	Large masses of tubercle on face and chin; smaller on extremities. Median nerves much enlarged. Sinuses and vessels of brain engorged. Hypostatic congestion of lungs. Spleen 9 oz., becoming diffluent. Malpighian bodies stand out like sago grains. Two splenculi. Blood fluid throughout body. Temperature in right pleural cavity 102·2°; in left 101·4°; in spleen 103·2; in liver 106°. (Necropsy two hours after death.) Gall-bladder full of bile. Bile in stomach and intestines. Slight redness in stomach. Femoral glands enlarged.
82	India	C	51	—	Mar. 7 1889	A	12	Ulceration and gangrene	None in kidney, spleen, liver, femoral gland, or median nerve	Body wasted. Gangrene of stump of right leg. Large ulcer over left outer malleolus. Symmetrical punched-out ulcers over buttocks. Median nerves thickened. Old deformity of fingers. Femoral glands enlarged. Pleuræ adherent. Spleen adherent to diaphragm. Kidneys 4 oz. Cortices filled with small cysts.
83	India	C	—	39	Mar. 30, 1889	T	5	Obstruction of larynx	Few in liver and femoral gland. None in spleen, median nerve, kidney, or larynx	Large masses of tubercle on face, hands, and feet. Epiglottis ulcerated; also vocal cords. Thickening and ulceration of mucous membrane of larynx; lumen blocked below cords. Spleen 10 oz. Femoral glands and median nerves enlarged.
84[2]	India	C	50	—	April 1, 1889	T	10	Addison's disease	Few in liver and femoral gland. None in adrenals, ganglia of aortic plexus, spleen, larynx, kidney, or median nerve	Body rather wasted. Nose sunken. Corneæ opaque. Skin dark, rough. No extra pigment in axilla or groins. Fingers thickened; commencing ulceration at tips. Scars on prepuce, with contraction of orifice. Increase of pigment on inner surface and on glans. Line of pigment on inner surface of lips and along gums. Ulceration and gangrene beginning in mouth. Median nerves thickened. Tip of epiglottis ulcerated. Pigment in mucous membrane above epiglottis. Ulceration and gangrene beginning here also. Uvula nearly sloughed away, and fauces ulcerated. Mucous membrane of larynx thickened and ulcerated. Pleuræ adherent in places. Several ounces of fluid in left pleura. Lungs rather œdematous at bases. Kidneys 8 oz.; some

										cysts. Old gumma on upper surface of liver, with some puckering. Adrenals lying in much fat; converted into sacs containing brown débris, and lined with yellow grains looking like minute tubercles. No trace of medullary substance. Aortic ganglia enlarged; dark red. Brain 38 oz.; healthy. Much dark brown pigment in pia mater over medulla and upper part of cord. Femoral glands enlarged.
85	India	C	24	—	April 20, 1889	M	7	Ankylostomiasis. Mixed kidney. Hæmorrhage after amputation	Few in larynx, femoral gland, median nerve. None in liver, testis, adrenal, kidney, or spleen	Body wasted, pale. Space between flaps of stump filled with dark blood-clot. Median nerves somewhat thickened. Right sciatic (which was stretched) shows some yellow degenerated fibres. Ulcer on right sole healed. Fingers thickened, ulcerated. Mucous membrane of larynx thickened. Brown discolouration and slight ulceration over vocal cords. Few pleuritic adhesions on right side. Kidneys 9 oz.; cortices blurred; capsules adherent. Right adrenal 60 grains; left 105 grains. Structure normal. Numerous ankylostomata and hæmorrhages in jejunum. Encysted hydrocele of right testis. Hard fibrous tissue encroaching on tubules, which are interspersed with brown pigment. Femoral glands enlarged.
86	Trinidad	B	24	—	April 28, 1889	T	16	Tuberculosis. Mixed kidney.	Few in median nerve. None in adrenal, lung, liver, kidney, spleen, or femoral gland	Body very wasted. Suppurating cervical and femoral glands. Median nerves enlarged. Healed ulceration of fingers. Nails grooved and brittle. Mass of small tubercles at left apex, causing puckering and adhesion. Tubercles generally diffused through right lung. Spleen 8 oz.; several small tubercles on and near surface. Kidneys 7 oz.; capsules adherent; cortices blurred, and gelatinous looking. Some staining with iodine. One or two yellow tubercles.
87	Trinidad	B	—	44	Aug. 4, 1889	A	?	Phthisis. Tuberculosis	In median nerve numerous deeply-stained cells with bacilli. No bacilli in lung, liver, spleen, kidney, or ovarian cyst	Body very wasted. Old spontaneous separation of fingers. Pleuræ adherent. Cavity at right apex, with consolidation round. (Aspiration here had given relief during life.) Grey tubercles generally diffused through left lung. Spleen 12 oz.; simple hypertrophy; contains numerous small yellow tubercles. Several similar tubercles in liver. Suppurating ovarian cyst, about size of two fists.

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[1] 'Lancet,' 1889.

[2] Ibid.

No.	Birth-place.	Colour.	Age. M.	Age. F.	Date of death.	Form of leprosy.	Duration in years.	Cause of death.	Distribution of bacilli.	Post-mortem appearances.
88	Trinidad	B	–	16	Sept. 7, 1889	T	4	Ulceration. Cardiac hypertrophy and dilatation	Numerous in femoral gland, liver, spleen (some in cells). Generally diffused in epiglottis. Few in kidney. None in heart or median nerve	Serpiginous ulceration on legs, beginning to be gangrenous. Superficial ulceration of hands. Median nerves greatly thickened above wrists; fusiform; individual fibrils much enlarged. Superficial ulceration of epiglottis and vocal cords. Right pleura adherent. Heart 10 oz.; walls of left ventricle greatly hypertrophied. Cavities dilated. Spleen 10 oz.; simple hypertrophy. Kidneys 9 oz.; congested; numerous small ecchymoses in cortices and beneath mucous membrane of pelves. Femoral glands much enlarged.
89	Trinidad	B	15	—	Nov. 16, 1889	M	4	Large white kidney	Few in median nerve. None in liver, kidney, spleen, testis, or femoral gland	Intense anæmia. Œdema of extremities. Most of tubercles absorbed. Scaly patches on lower extremities. Finger-nails broken. Median nerves thickened. Two large patches of pleuro-pneumonia at left base. A good deal of effusion in both pleuræ. Upper part of right lung sodden and fleshy. Spleen 11 oz.; becoming diffluent. Kidneys 14 oz.; large white; mottled on surfaces. A good deal of fluid in abdomen. Femoral glands slightly enlarged.
90	India	C	43	—	Nov. 18, 1889	M	6	Granular kidney. Tuberculosis	Few in liver, median nerve, and lung. None in spleen, kidney, testis, or femoral gland	Body wasted. Skin rough and dry. Many tubercles absorbed. Fingers and toes ulcerated. Pleuræ adherent in places. Patch of consolidation at left apex, with small cavities and a few tubercles. Spleen 8 oz.; firm; several small translucent tubercles. Kidneys 5 oz.; cortices thinned; capsules adherent in places. One small yellow tubercle in right cortex. Many ankylostomata in duodenum and jejunum; no hæmorrhages seen. Femoral and mesenteric glands enlarged. Median nerves thickened; brown; individual fibres enlarged.
91	Trinidad	B	—	70	Dec. 13, 1889	A	19	Atheroma. Cerebral thrombosis	None in cortex, pons, median nerve, or atheromatous cerebral artery	Fingers contracted. Old separation of toes. Median nerves thickened. Slight adhesions of pleuræ. Kidneys 4 oz.; granular. Arteries of circle of Willis very atheromatous; thrombosis in some of them.

92[1]	India	C	35	—	Dec. 20, 1889	A	13	Mixed kidney. Dysentery	None in spleen, liver, adrenal, median nerve, femoral gland, kidney, or ulcer of large intestine	Body wasted. Median nerves atrophied. Stump of thigh firmly healed. A few pleuritic adhesions. Spleen 24 oz.; simple hypertrophy. Kidneys 7 oz.; left capsule very adherent; cortex thinned and blurred; right less affected. Large intestine intensely congested; small ulcers in places.
93	India	C	—	29	Dec. 25, 1889	T	7	Large white kidney	None in liver, kidney, tongue, larynx, femoral gland, median nerve	Body wasted. Suppurating axillary, femoral, and inguinal glands. Ulcerating tubercles on hands. Many tubercles absorbed. Median nerves thickened. Thickening and ulceration of mucous membrane over epiglottis, vocal cords, and ventricles of larynx. Mouth much contracted from ulceration. Tongue thickened, scarred. A good deal of fluid in pleuræ and peritoneum. Spleen 6 oz.; a little thickening of capsule. Kidneys 10 oz.; large white.
94	Tobago	B	—	14	Feb. 12, 1890	T	5	Mixed kidney. Tuberculosis	None in liver, spleen, kidney, lung, femoral gland, median nerve, tubercle of intestine, or of diaphragm	Body wasted. Most of tubercles absorbed. Median nerves atrophied. Slight puckering and adhesion at apices of lungs; no tubercles found. Liver tough; contains a few small pale yellow tubercles. Kidneys 7 oz.; pale; surface rough; contain a few small tubercles. Spleen 8 oz.; hard; old scars on upper surface; contains a few small tubercles. Peritoneum over intestines and diaphragm covered with pale tubercles the size of small shot. Confluent in many places. Several pints of clear fluid in abdomen. Femoral glands enlarged.
95	Trinidad	B	41	—	Feb. 20, 1890	M	10	Obstruction of larynx	Few in liver. None in larynx, spleen, kidney, femoral gland, or median nerve	Most of tubercles absorbed. Median nerves much enlarged. Corneæ opaque; tuberculated. Tongue thickened. Epiglottis tuberculated and rounded; almost globular. Much ulceration of larynx. Vocal cords destroyed. A little fluid in pleuræ. Spleen 15 oz.; hypertrophied. Femoral glands about 2 inches in diameter.
96	Barbados	C	58	—	Feb. 28, 1890	A	—	Tuberculosis. Gangrene	Numerous in median nerve. None in adrenal, testis, liver, spleen, kidney, lung, or femoral gland	Body wasted. Gangrene of right foot and swelling of right lower extremity. Femoral glands enlarged. Median nerves somewhat enlarged. Both lungs full of small grey tubercles. Spleen 6 oz.; numerous small yellow and grey tubercles. Kidneys 8 oz.; one small tubercle.

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[1] 'Brit. Med. Journ.,' 1885 and 1890.

No.	Birth-place.	Colour.	Age.		Date of death.	Form of leprosy.	Duration in years.	Cause of death.	Distribution of bacilli.	Post-mortem appearances.
			M.	F.						
97	Trinidad	C	12	—	Mar. 11, 1890	T	7	Tuberculosis	Numerous in testis, lung, and kidney. Few in spleen and liver. None in adrenal, femoral gland, and median nerve	Body very wasted. Nearly all tubercles absorbed. Both lungs full of small tubercles. Pleuræ adherent. Spleen 7 oz.; filled with small tubercles. A few in liver. Testes only partly developed. Femoral glands slightly enlarged.
98	Trinidad	C	—	32	May 9, 1890	A	20	Mixed kidney	None in liver, kidney, spleen, femoral gland, or median nerve	Œdema of whole body. Old separation of nearly all fingers. Ulceration and dead bone in left hand and left foot. Median nerves somewhat thickened. About a pint of fluid in left pleura; lung sodden. Kidneys 15 oz.; large white; capsules adherent in places. Many pints of flaky fluid in abdomen.
99	India	C	63	—	May 10, 1890	A	4	Abscess of ankle. Hydronephrosis. Ankylostomiasis	None in liver, kidney, spleen, testis, femoral gland, median nerve, skin	Body wasted. Median nerves enlarged. Bones of left ankle carious; cartilages gone; gangrene commencing; pus burrowing along tendons. Small nodule of atheroma on one aortic valve, and few small patches on aorta. Kidneys 10 oz.; contain a few cysts; pelves much dilated and filled with urine; pyramids encroached on. Bladder extremely sacculated. Phimosis and scars on prepuce. Numerous ankylostomata in duodenum and jejunum. Femoral glands enlarged.
100	Trinidad	C	—	36	May 28, 1890	M	9	Mixed kidney	None in liver, spleen, kidney, median nerve, or femoral gland	Stump of amputated right leg nearly healed. One femoral gland beginning to caseate. Median nerves slightly enlarged. Some adhesions of pleura. Heart 7 oz.; very flabby; muscle anæmic. Spleen 10 oz.; hypertrophied. Kidneys 6 oz.; capsules strip easily, but cortices are thinned and blurred. Liver pale; fatty.
101	India	C	50	—	June 11, 1890	A	5	Atheroma and hypertrophy of heart	None in liver, spleen, kidney, median nerve, femoral gland, testis, pons, or cerebral cortex	Median nerves slightly enlarged. Femoral glands enlarged. Heart 13 oz.; greatly hypertrophied, especially left ventricle. Patches of atheroma on aorta, mitral valve, and one of aortic valves. Mitral orifice contracted. Spleen 12 oz.; hypertrophied.

102	Africa	B	—	65	June 12, 1890	A	5	Gangrene of lung	None in liver, spleen, kidney, median nerve, femoral gland, or pneumonic lung	Pale patches still very evident on chest and abdomen. Ulceration and destruction of bone in hands and feet. Median nerves thickened. Femoral glands enlarged. Upper lobe of right lung gangrenous; liquefied. Small patch of consolidation at left apex, beginning to break down. Pleuræ adherent. Liver extremely fatty. Spleen 9 oz.; becoming diffluent. Kidneys 6 oz.; fatty.
103	Trinidad	B	—	34	June 14, 1890	M	4	Mixed kidney	None in liver, spleen, kidney, femoral gland, median nerve, pons, cerebral cortex, or spinal cord	Body wasted. Kidneys 8 oz.; cortices blurred; capsules partly adherent. Other viscera normal.
104	Trinidad	C	21	—	June 15, 1890	M	7	Malarial fever	None in liver, spleen, kidney, femoral gland, or median nerve	Body well nourished; plenty of subcutaneous fat. Blood fluid throughout body; hardly any clotting. Lungs œdematous. Spleen 13 oz.; hypertrophied; becoming diffluent. Median nerves thickened. Femoral glands enlarged.
105	Born at sea	W	38	—	Aug. 6, 1890	M	15	Phthisis	Numerous in spleen, liver, kidney, and femoral gland. None in larynx, median nerve, testis, or lung	Body wasted. Gangrenous ulcers on hands, legs, and feet. Suppurating glands in neck. Old separation of phalanges of fingers. Median nerves much thickened above wrists. Thickening and ulceration of epiglottis, ary-epiglottic folds, vocal cords, and larynx below cords. Tracheotomy incision healthy; ¼ inch long, and 1½ inches below vocal cords. (Tracheotomy 4 months ago.) Pleuræ adherent. Both lungs full of grey miliary tubercles. These are beginning to caseate and break down at right apex. Lungs solid in places from agglomeration of tubercles. Liver tough; cirrhotic; lessened in size. Spleen 21 oz.; hypertrophied. Kidneys 14 oz.; lardaceous. A few ounces of straw-coloured fluid in abdomen. Femoral glands enlarged.
106	Trinidad	B	61	—	Aug. 15, 1890	M	15	Pleuro-pneumonia	Numerous in liver. Few in femoral gland, kidney, and spleen. None in testis, median nerve, or lung	Body well nourished. Old loss of bone and contraction in hands and feet. Remains of tubercles on face. Median nerves much thickened. Femoral glands enlarged. Heart 13 oz.; hypertrophied. Right pleura contains about a pint of dirty purulent liquid and some recent flaky lymph. Red hepatisation of right lung, except apex. Spleen 11 oz.; hypertrophied. Kidneys 10 oz.; cystic.

No.	Birth-place.	Colour.	Age.		Date of death.	Form of leprosy.	Duration in years.	Cause of death.	Distribution of bacilli.	Post-mortem appearances.
			M.	F.						
107	Trinidad	B	23	—	Sept. 4, 1890	T	2	Tuberculosis. Pericarditis	Numerous in median nerve. None in heart, testis, lung, femoral gland, kidney, spleen, liver, or tubercle of skin	Body wasted. Still many tubercles on face and extremities. Median nerves enlarged; individual fibres thickened and gelatinous. Femoral glands enlarged. Both lungs full of small grey tubercles. Left pleura adherent towards front of chest; at back contains about a pint of blood-stained fluid. Left lung very œdematous. Anterior mediastinal glands caseating. Pericardium filled with layers of partly organised lymph. Total thickness of layers about ¾ inch. Stratum nearest heart bright yellow; about ¼ inch thick; becoming calcareous in places. Heart pale and flabby; cavities dilated; valves healthy; 16 oz. (with thickened lymph). Spleen 15 oz.; hypertrophied; a few small yellow tubercles.
108	India	C	40	—	Sept. 5, 1890	A	—	Ankylostomiasis	None in liver, kidney, spleen, femoral gland, or median nerve	Œdema of extremities. Old separation of fingers and toes. Plenty of bright yellow subcutaneous fat. Pleuræ adherent. Liver 27 oz.; fatty; very anæmic. Quantities of ankylostomata in duodenum, fewer in jejunum, very few in ileum. Much dark tarry blood in bowel down to ileo-cæcal valve.
109	India	C	40	—	Sept. 18, 1890	M	3	Tuberculosis. Ankylostomiasis	None in liver, spleen, kidney, testis, femoral gland, or median nerve	Body extremely wasted. Right femoral glands caseous. Left have suppurated away through numerous sinuses. Left foot swollen. Lungs full of grey tubercles; cavity commencing at right apex. Old adhesions between upper surface of liver and diaphragm. Mesenteric glands caseating. Numerous miliary tubercles on peritoneum. Numerous ankylostomata in small intestine.

A THEORY OF SMALLPOX.

By R. A. BIRDWOOD, M.D.

(Communicated by J. W. WASHBOURN, M.D.)

The manifestations of smallpox are, I think, more readily explained on the supposition, that the disease is a surface mycosis than that it is a blood zymosis. The editors of the 'Guy's Hospital Gazette' kindly gave me the opportunity of stating this proposition and the evidence supporting it; this paper is intended to be a more complete exposition of the articles in the 'Gazette,' addressed to a more extended circle of Guy's men and to the medical profession generally. I trust that the opportunities I have had at the Smallpox Hospital for the observation of this disease are a sufficient justification for submitting my theory to the touch of pathological criticism.

Smallpox is, as far as we known, communicated by means of particles. These exist in various parts of the body and are diffused in the surrounding air.

Workers in dust-laden atmospheres have the dust deposited on their surfaces in varying quantities. The alimentary and respiratory tracts above the level of the larynx will be very thickly covered, but in the œsophagus we rarely find any. On the skin the dust is deposited in definite patterns varying with the conditions of exposure to it. The space between the body and the clothes is well ventilated. The temperature of the body being higher than that of the air, its heat is communicated to the clothes and to the mass of air between the body and them. This air expands and escapes. As it escapes from within the space it will be replaced by air from without. If this air carries particles capable of adhering to the surface of the body the dust will be deposited on different parts in varying densities, and will adhere where deposited, according to the nature of the surface. The best-aired surfaces will have the most, the worst-aired the least; more will adhere to damp surfaces than to dry; more on rough than on smooth. The site of a garter, boot-lace, brace, or belt is marked by an accumulation of dust, the pattern of the obstruction being mapped out on the surface. More adheres to the back than to the chest, for the clothes fit more closely to the front of the trunk than to the back, especially along the spines. The most striking peculiarity, however, is that the conjunctivæ remain clear in the midst of all the dirt. This is well seen, in the case of a coal-heaver, where the white sclerotics contrast with the blackened skin. If we could substitute, for each particle of coal-dust, a drop of blistering fluid or croton oil, an eruption, similar in distribution to the eruption of smallpox, would result. If the air was laden with living spores, capable of proliferating on, and vegetating in, the human skin and mucous surfaces, and if during such growth pustules were formed, this would account for the distribution of the smallpox eruption. Whatever determines the deposition of dust on the bodies of workers in dust-laden atmospheres equally determines the deposition of infectious air-carried material on the persons exposed to infection. At the time of infection with smallpox the spores of the *Micrococcus variolæ* are deposited on the surfaces of the body, according to laws regulating the deposition of any other air-carried dust.

The surface lesions, resulting from the growth of vegetable parasites, are represented by ringworm and thrush. In an ill-ventilated schoolroom air-carried spores of Tricophyton tonsurans may be regarded as the infectious material. The following passage is taken from Mr. Waren Tay's translation of Hebra and Kaposi's 'Diseases of the Skin;'

published by the New Sydenham Society in 1880, vol. v, p. 215: "In the first place the fungus takes root in the superficial epidermic layers, after the most superficial layers of all have been removed by maceration, or some other mechanical means. The fungus elements act as a foreign body, irritating the subjacent papillæ. These become hyperæmic. The part becomes congested and appears as a red spot. If the patient's skin, or that particular portion of skin, is specially irritable, the hyperæmia increases to such a degree that serous exudation follows, and then, if the exudation be moderate, the epidermis is loosened, and then, after evaporation of the serum, lifted up in scales, or, if more abundant, is raised into vesicles. The fungus is lifted up with the epidermis, for it never goes deeper and never penetrates into the corium. Thus it happens that, at the spot originally diseased, the fungus is eliminated by the very exudative process started by itself, and here, therefore, a spontaneous cure must result. On the other hand, the parasite grows at the periphery of the original focus, in the layers of the adjacent epidermis where it excites the same symptoms as in the first part attacked. The peripheral advance of the morbid phenomena and the subsequent involution following, in the same direction, are thus easily comprehended." Again on p. 201: "Occasionally the vesicles of *Tinea tonsurans vesiculosa* are developed in considerable numbers and of an unusual size, that of a pin's head or larger. In such a case and corresponding to the greater amount of exudation, the redness and swelling of the skin will be moderately severe. The larger quantity of fluid exuded under the circumstances, dries up into thick, yellow, gummy crusts, so that the appearance of the eruption resembles that of a very moist, acute eczema. Fever may also accompany such an eruption if it is spread over a large part of the trunk." Also on p. 216: "Just as in the case of the growth in the epidermic layers of the surface of the skin, the fungus, by its accumulation in bulk in the epidermic layers of the hair follicle, may excite irritation and the formation of tubercles and pustules, producing, in fact, the symptoms of *Sycosis parasitaria*, or of *Herpes tonsurans pustulosus*." It will be observed that an undoubted mycotic process produces all the forms of skin lesion that constitute a pock in smallpox; it is merely a question of degree.

The growth of a vegetable parasite on the mucous membrane produces an altogether different eruption. The following passage is from Dr. Fagge's 'Principles and Practice of Medicine,' 1886, vol. ii, p. 99. "The earliest indication that thrush is setting in, is a change in the mucous membrane lining the cheeks and other parts of the mouth; it becomes redder than natural, hot and painful. Soon a number of minute milk-white spots appear upon its surface. These rapidly increase in size, and run together; and in a day or two, the whole surface may be covered with a pearly uniform adherent layer. At first, there is some difficulty in detaching the white material, but after a time it becomes quite loose, and can be peeled off in large flakes without any bleeding. Microscopically it consists, partly of layers of squamous epithelium, partly of the spores and mycelium of a fungus. The fungus is usually known as the *Oidium albicans.*" The smallpox eruption on the respiratory surface and in the mouth, resembles the eruption above described. It differs from a pock on the skin in that whatever is exuded from the blood vessels is not collected to form a vesicle or a pustule. This is explained by the difference of moisture in the two situations, and by the fact that the cuticle is tougher than the most superficial layer of a mucous membrane. The incubation period may be accounted for as the time during which the micro-organism is living on the non-irritable superficial epithelium. When it has grown through this layer and begun to invade the capillary walls, the injury to those vessels is followed by the inflammatory reactions resulting in a pock on the skin, or a patch on the mucous membranes. These two lesions only occur at sites where the spores could have been carried by the air; they are not found in positions inaccessible to it. At the end of the period of incubation the initial illness of smallpox supervenes, sometimes this is accompanied by a rash or a purpura. Occasionally the affection of the mucous membrane is visible at this early stage, when the pocks on the skin cannot be seen or felt. The rash and the purpura are both of them evidences of vascular changes, as are also the petechiæ, which may be seen in synovial and serous membranes and in all the tissues, but nothing resembling a pock or a patch occurs there. Later on, the characteristic eruptions of smallpox are apparent; these are inflammations of the skin or acute dermatitis, and of the upper respiratory surfaces, are due, as I contend, to the local growth of the microorganism of smallpox. Regarding now these two surfaces as viscera having important excretory functions, implication of them probably means arrest of their functions and consequent retention in the blood of the toxic substances which would otherwise be excreted from their surfaces. It is to this poison that the symptoms of the initial illness, including rash and purpura, are due. The rash or hæmorrhage does not indicate a severe attack of smallpox; for the present it may be said that it can only be explained by some idiosyncrasy on the part of the patient. Just as some foods or drugs are followed by urticaria, petechiæ, or hæmaturia in some persons and not in others; as when quinine produces hæmaturia, or iodide of potassium leads to a petechial eruption, or the administration of salicylate of soda is attended with epistaxis, retinal hæmorrhage, hæmaturia, and ecchymosis of the pelves of kidneys; this is evidence of some peculiarity on the part of the patient, but not necessarily evidence of the growth of an organism in the blood.

The fever of maturation of smallpox calls for no special comment. Indeed the symptoms of smallpox, from the beginning to the end, may be likened, stage by stage, to those resulting from a scald or burn of equal extent. The attack of smallpox may be considered to end with the drying of the pustules; sequlæ being brought about by one of the wound-infecting diseases complicating the normal progress of the disease.

It therefore seems to me probable that the pock on the skin and the patch on the mucous membrane are the consequence of the irritation caused by the proliferation of a spore at the spot where it has adhered. It has been stated that this formation of a pock has been seen in the fœtus and the newly-born child, on serous and synovial membranes. I have looked for it in these places, but I have not seen it. Petechiæ are common even in the serous membranes of the fœtus whose mother has not had hæmorrhagic smallpox, but has been suffering from the ordinary form of the disease.

The trachea and bronchi of sufferers from smallpox are covered with the eruption, the œsophagus is free from it. The skin of the face suffers severely, the conjunctiva hardly at all. On the pharynx the part in the air stream from the nose to the larynx may be crowded with patches, the part below the air stream may have none. If the usually-accepted zymotic theory is true, the œsophagus should be as much attacked as the trachea; the lower part of the pharynx as severely as the upper; the conjunctiva as abundantly as the face. Under the adherent part of the nail pocks do not occur; if the disease is a zymosis they ought to be there. We might certainly expect some lesion on the serous and synovial membranes, but excluding petechiæ, there are none early in the disease. When there is any morbid condition in these parts it is generally late in the disease, after general septic infection has been possible.

The distribution of the variolous eruption on the skin also corresponds closely with the distribution of dust on the bodies of workers in dust-laden atmospheres. There is most on the uncovered surfaces, more on the back of the trunk than on the front. Warm moist surfaces have more than cold dry parts. On the feet the distribution is like that of dust. It is, however, in exceptional instances of the distribution of the eruption that the demonstration becomes convincing. If, soon after infection, the patient happens to have washed the hands or face, there will be less there than on other parts. A girl, aged ten years, was admitted with an abundant eruption of confluent vesicles on her face, arms and legs. She had hardly any on her trunk. The boundaries between the parts covered with eruption, and those free from it were sharply defined. The history of this girl's infection was this:—A boy, supposed to have had chicken-pox, was moved out of a bed, and she was put into it without the sheets having been changed. Shortly afterwards she complained that the bed was full of bread-crumbs. On directing attention to the matter she was removed from that bed. I suppose the bread-crumbs were probably scabs from the boy patient, whose illness had been mistaken for chicken-pox, but was really smallpox. This girl was in the habit of wearing a woollen sleeveless jersey, instead of a night-gown. Close-fitting woollen garments being efficient filters for air, protect the surface covered by them. She had a peculiar habit: before jumping into bed she used to take hold of the back and front of her jersey at its lower margin and hold the two in one hand between the legs. She retained hold after she was in bed and during sleep. Now when she did so, the edges of the jersey at the neck, arms, and groins, fitted the boundaries between the affected parts and those that had escaped the eruption. It seems clear that she had been infected by going into that bed, and that the jersey had prevented the spores from being deposited on the trunk.

A second case in point is that of a bricklayer, aged 38 years, who had been out of work for some weeks past. He had been walking to London, occasionally resting at a casual ward. He stated that he had been vaccinated in infancy and he thought once since, when he was fourteen years old. The scars on his arms were indistinct. At one workhouse he stopped two nights. On the first night a man in the same room was very ill and had "spots." This man was removed. In reply to inquiries, the porter stated that the "man had spots on him." On the second night the bricklayer was given the rug the sick man had used, to cover himself with. Being tired, he slept soundly in it. On admission to the Smallpox Hospital, he had a discrete vesicular eruption on his face, only ten spots on the forehead, and much less on his hands and face than on his body. He had extensive patches of confluent eruption, exceptionally distributed on his arms. On the right arm it began about two inches above the elbow and extended two thirds round the arm; it continued down the extensor surface of the forearm to about three inches above the wrist; then there was a space free from eruption, and a confluent patch on the back of the hand. On the left arm the eruption began at the same place and extended down the extensor surface of the forearm to the back of the hand, half way to the fingers. This man is in the habit of sleeping on his back in his underclothes, with the sleeves tucked in above the elbows, his forearms crossed over his chest, the left forearm resting on the right, and he has his hands partly embedded in his axillæ. When he is in this attitude, a rug covering him would be in contact with the parts on which the confluent pocks were situated. The portion of the right forearm covered by the left had escaped.

The reason why so many unvaccinated babies have discrete smallpox probably is that as they are bathed so often, the spores are washed off soon after being deposited.

It is usual for the eruption to be far more abundant on the back than on the front of the trunk. A man had a severe and abundant eruption on the front of his body, and but few discrete pocks on his back. He was infected in a hospital. During and for weeks before infection he had been confined to bed and kept on his back. In this man's case there was another unusual distribution of the eruption which is usually confluent on inflamed parts of the skin. He had several cicatrising and granulating wounds, surrounded by highly vascular zones; these were the remains of pyæmic abscesses. The wounds were covered with boracic lint and wood-wool; at the time of his infection they would have been dressed antiseptically. In the neighbourhood of these sores there was an absence of pocks. The dressings had prevented the spores from falling on the skin.

The amount of eruption of pocks is determined by the circumstances of exposure to infection. Few smallpox patients can give any satisfactory account of the circumstances of their infection. With those who can it is evident that the number of pocks and the appearance of successive crops of pocks, are dependent on the length of exposure and proximity to the source of infection. The disturbing factors are individual insusceptibility, congenital or acquired, personal cleanliness, and clothing. Insusceptibility, complete or partial, is the result of a previous attack of smallpox or of vaccination. It is said that some persons are insusceptible without having had either of these diseases, and that others owe their immunity to their mothers having had smallpox during their pregnancy with that child.

All these modifying causes being eliminated, and the mode of infection being known, it will be found that length of exposure to infection has a great deal to do with the severity or mildness of the attack. In smallpox there is a sequence in the eruption, and this may be divided into two stages In the first the eruption appears on certain parts of the body in a definite order, beginning with the face or wrists and ending with the legs. The difference in time between the outbreak on most and least favoured sites is two days or so. The second stage is quite different, and consists of additional crops of pocks on the same sites as before, and developing in the same order, but fresh crops keep on coming out for three or four days. Successive sowings of spores result in successive crops of pocks. If some patients happen to have been exposed for a short time only, the eruption on that particular part will all or almost all appear on one day. If others are exposed from day to day, crops of pocks continue to come out for three or four days. It is as if successive sowings resulted in papules after the lapse of a constant period on the same soil, at varying periods on different soils; so that if the patient was exposed to infection on the 1st of the month, papules would appear on the forehead on the 14th, on the legs on the 16th, but if he was daily exposed from the 1st to the 4th and after, then the papules would keep on coming out from the 14th to the 17th on the face, and from the 16th to the 19th on the feet.

A child told her schoolfellow that she had just recovered from smallpox, and pointed out some adherent scabs. About a fortnight later this schoolfellow fell ill, and her illness was diagnosed as chicken-pox. An elder sister, vaccinated in infancy, attended her during her illness. She was admitted into hospital with an abundant eruption of smallpox. An indefinite number of instances might be given in which short exposure to infection, such as calling to make inquiries about a relative or friend ill of smallpox, or remaining in an infectious place for a few minutes, has resulted in discrete eruptions, even where there is no question of protection by vaccination or otherwise. This was well known formerly, and was made use of by the people of this country till "wilful exposure to variolous matter" was made a crime. The belief was that short exposures to smallpox infection produced a mild attack of the disease. On the other hand, such an exposure as that of a nurse, unprotected by the neglect of re-vaccination, in charge of a smallpox patient, is almost certain to lead to a confluent attack. The bedfellow of an undiagnosed case invariably has a severe attack, whilst the other children have a discrete eruption. Or we may have such an outbreak as this which occurred at a well known commercial establishment. Several young men employed there had smallpox about the same time. Their exposure to infection was so slight that not one of them had any knowledge how the disease was communicated. All of them had very few pocks each, but in two different families they communicated confluent disease to their younger brother or sister with whom they had been intimately associated, but discrete to the other members of the family. All happened to have been vaccinated in infancy; it was the younger ones who had the severe illness, and they presumably were better protected, as they had been more recently vaccinated. Where all the attendant circumstances of infection happen to be known, and modifying conditions can be excluded, the converse also holds—that is, short exposures do not induce confluent smallpox; long exposures do not lead to the discrete variety.

The phenomena of inoculated smallpox apparently present an obstacle to the acceptance of the main contention. They really, however, support the theory. The local and general eruptions of inoculated smallpox may be likened to the instances of concurrent smallpox and vaccination that are now comparatively frequently seen. On the occurrence of smallpox in a house the other members of the family would probably be re-vaccinated; or, new members of the staff joining at a smallpox hospital would be re-vaccinated before beginning their duties. The re-vaccination may be successful, but having been exposed to smallpox infection, that disease also develops. We may suppose that the person to be inoculated was at the same time exposed to infection. The other day a man aged sixty-two told me that he was inoculated when seven or eight years old. A string of boys and girls went into a room at a public-house. There was a smallpox patient in that room; a woman did the inoculation with a needle, and in one place only. He himself had from a dozen to twenty pocks of the general eruption; but he recollected a woman inoculating herself and three of her children with lymph from another of her own children. The mother and the three inoculated died of smallpox. By removing the cuticle when we inoculate we place the spores in contact with injured capillaries, reaction begins at once, that is, a papule can be seen and felt next day, perhaps. Accidentally inoculated smallpox is occasionally admitted into the hospital; it is usually the result of being scratched whilst attending a relative ill at home with smallpox. It is then common to find a pustule or two at the site of a surface injury on patients whose general eruption is papular or vesicular. A similar result may be produced by vaccine lymph, if at the time of vaccination some is smeared in the neighbourhood of the injured surface. An annular vesicle on unbroken skin often surrounds the scab of the pustule that formed at the injured site; the scab being the local pock, the ring of vesicles the representative of the general eruption.

In both inoculated smallpox and concurrent smallpox and vaccination, the general eruption may be as abundant as in the most severe confluent smallpox. It is all a question of exposure. In vaccination whether one is to have one pock or a thousand depends on the number of inoculations done, that is, on the soil prepared and the seed sown. If two vaccinations are done at an interval of a few days, both the operations may result in a crop of unmodified pocks being produced at the respective sites. This is analogous to the successive crops of pocks so often noticed in severe smallpox, and throws light on the causation of the successive crops. As marked a difference may be seen in the ages of the two patches of pocks on the same arm, the result of vaccinations at intervals of days, as may be seen between successive crops of pocks on a part in confluent smallpox, or even as between the local and general eruptions of inoculated smallpox. In the latter cases, the older group would result from spores placed on an injured surface, the younger from spores on an uninjured surface. Similarly, vaccination after smallpox infection may result in a pock if done at any time preceding vesiculation. The manifestations of the disease here recorded are incomprehensible on the zymotic theory: they are readily accounted for on the supposition that smallpox is primarily an inflammation of the skin and certain mucous membranes resulting from the local growth of the micro-organism of smallpox. Not only may smallpox and vaccination attack a person at the same time; they may also occur on patients suffering from other febrile diseases, as has been proved here by the successful vaccination of patients sent to a smallpox hospital, suffering from scarlatina, measles, chicken-pox, or typhoid. The vaccinations result in typical vesicles. It has also often been observed that sufferers from other febrile diseases may be subject to smallpox at the same time.

That micro-organisms are present in the skin during the period of incubation is proved by a reference to Fagge, vol. i, p. 25. A case "occurred at the Charité Hospital, of Berlin, in which variola was conveyed to a patient by the operation of skin grafting, the graft having been taken from a person who happened to be in the incubation stage of the disease. This seems to show that from a very early period the whole of the skin contains the virus."

It has been my intention to avoid burdening this article by a recital of cases in support of the arguments, as by doing so the paper would have been very much lengthened. When I have quoted cases it has been my object in so doing to present the point more clearly than by a laboured description. The theory itself rests on a basis of facts; facts too numerous to quote in detail; facts that can only be profitably gathered at the bedside of the patient. It is to be hoped that the time is near when the student of pathology will spend some part of his working day beside the smallpox patient. If the correctness of the observations recorded here is admitted, the usual zymotic theory of smallpox will not explain the phenomena of it. I have ventured to propound an alternative theory, one full of hope to the workers at preventive medicine.

SEVEN CASES

OF

INTRA-SPINAL HÆMORRHAGE

(HÆMATOMYELIA).

By ALFRED PARKIN, M.S., M.D.

Cases of intra-spinal hæmorrhage are so seldom recorded and so rarely seen post mortem, that it seems worth while to give details of four cases which I watched throughout, and which I had the opportunity of verifying after death.

Besides these, I have included three cases which are recorded in the 'Surgical Reports,' one of which I saw during life.

From the number of cases which occurred during the time I was Surgical Registrar, I am inclined to think that this affection is by no means so uncommon as is at present supposed; and that greater refinements in diagnosis, and more careful investigation post mortem, will reveal many unsuspected cases.

My thanks are due to several of the staff for their kind permission to publish reports of cases under their care.

Case 1. *Fracture of sixth cervical vertebra (no displacement); intra-spinal hæmorrhage.*—Henry S—, æt. 33, admitted October 25th, 1890, under the care of Mr. Lane.

History of accident.—Patient was repairing a chimney when he over-balanced himself and fell a distance of forty feet on to a garden wall; he says he fell on to his stomach. Some bricks falling at the same time struck him upon the back.

On admission (7.30 a.m.).—Patient was quite conscious and could answer questions, but his replies were somewhat hesitating. Feet everted, legs could not be moved voluntarily, and the paraplegia extended up to and involved the arms. He could raise his hands to his head, but could not move his fingers; very slight pressure on the arms was sufficient to prevent any movement at all.

Abdomen bulged in inspiration, abdominal muscles quite flaccid; absolutely no movement of thorax.

Doubtful plantar reflex on left side; that on right side absent. Knee-jerks absent. No ankle-clonus. Cremasteric, abdominal, and epigastric reflexes absent.

Anæsthesia complete below level of second intercostal space; no ring of hyperæsthesia.

Patient says his arms feel numb, but he localises sensations in them very readily; sensation in the face and neck very good; no facial paralysis. Pupils equal and react to light. Priapism marked.

The spines of sixth and seventh cervical vertebræ can be easily felt, but those above cannot be distinguished. Pressure on the sixth and seventh spines gives rise to a good deal of pain, but no crepitus felt. No irregularity to be made out in the line of the cervical transverse processes.

Urine (drawn off by catheter), sp. gr. 1030. No albumen, sugar, or blood. P.=90. Resp.=20. Temp. 98°.

October 26th.—No change in reflexes, except that the slight left plantar reflex has disappeared. No alteration in sensation or in power of movement. Hands kept in prone position, fingers flexed. Passive supination for 5° causes much pain. Patient complains of dull aching pains in both arms.

Abdomen has gradually become more and more distended; intestinal coils are visible. Urine drawn off by a catheter; no incontinence. Priapism still present. A little cough has developed and a few râles can be heard in front of chest. Pupils have become much contracted.

11.15 p.m., abdominal distension extreme. No movement of abdomen in inspiration, breathing carried on apparently only by sterno-mastoids and scaleni. Profuse sweating. Pupils contracted to a minute size.

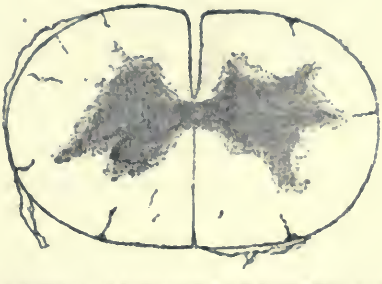
At 10 a.m. temp. was 102·6,° at 6 p.m. 104,° and at

11 p.m. 107·6°. Respiration failed, and patient died cyanosed at 11.40 p.m.

Beyond an injection of morphia on the night of the 25th, and keeping the head fixed by sand-pillows, no treatment was adopted.

Post-mortem.—On examination of the front of cervical spine there was seen to be no irregularity over the front of the bodies of the vertebræ, and the anterior common ligament was everywhere intact. The anterior part of the sixth cervical transverse process was found loose on the right side, and there was a little effused blood in the longus colli at this spot. The right lamina of the sixth cervical vertebra was broken across transversely, but there was no displacement of the spines. There was a fracture of the sixth cervical spine at its junction with the laminæ. On further examining the bodies, that of the sixth cervical was seen to be divided into two parts by a horizontal fracture near to the attachment of the fibro-cartilage below, but there was no displacement. No intra- or extra-meningeal hæmorrhage, but the cord felt tense opposite the fracture.

After removal of the cord there was decided softening for about 2¼ inches; on cutting it across extensive central hæmorrhage was seen breaking up the cord substance but nowhere reaching the surface of the cord. The destruction was greatest opposite the level of the fracture, where there was only a rim of apparently healthy white matter; above this the cord was damaged in the centre (grey matter) and in the right lateral column; below, the hæmorrhage was



This figure represents an enlarged view of the naked-eye appearance of the section of the cord.

confined to the grey matter and left lateral column. The length of the cord damaged would correspond to the bodies of the fifth, sixth, and seventh vertebræ. Considerable atheroma of whole of aorta.

The points of special interest in this case are the complete paraplegia up to the arms, complete loss of sensation below second intercostal space, absence of all superficial and deep reflexes, extreme myosis, rapid rise of temperature to 107·6°, and severe abdominal distension.

The mode of production of the injury is not quite clear; the patient said he fell on his stomach, and that some bricks fell on his back; one of these might have struck him in the cervico-dorsal region. This view is supported by the comminuted fracture of the sixth cervical vertebra unaccompanied by displacement.

CASE 2. *Old spinal caries; dislocation of cervical spine; intra-spinal hæmorrhage.*—W. W—, æt. 65, admitted January 26th, 1891, under the care of Mr. Durham.

History of accident.—Patient was mounting a ladder in the stable, when he missed his footing and fell a distance of six feet on to his head. He was picked up unconscious, and brought straight to the hospital.

On admission.—He was much collapsed, and quite unable to walk; he could just move his head and arms a little. Speech was low and there seemed to be some difficulty in articulation. Breathing jerky and entirely abdominal. Plantar reflexes marked. No ankle-clonus, but a distinct "front-tap" contraction. There was an absence of definite knee-jerks, but a slight contraction of the quadriceps extensor could be seen on striking the ligamentum patellæ.

Abdominal, epigastric, and cremasteric reflexes all absent. Priapism present. Urine drawn off by catheter. Complete paraplegia up to arms; the patient can just flex each forearm for a few degrees. Complete anæsthesia below a horizontal line drawn at the level of the second rib at its junction with sternum. No region of hyperæsthesia.

No paralysis of head or face, the head can be rotated from side to side. Pupils contracted markedly. A distinct bony click was obtained at about the level of the sixth or seventh cervical vertebra. There was a contused wound on the forehead, apparently where the patient struck the ground in falling.

January 27th.—Condition much the same. Patient can still move his forearms a very little; he complains of great pain in his neck. Good plantar reflexes and knee-jerks. No ankle-clonus. There are marked movements (reflex?) of the legs and thighs, which are drawn up occasionally and then extended. The patient has, however, no knowledge of these; they give rise, apparently, to no sensations whatsoever.

Pupils very contracted, but still react to light. Chest full of mucous râles.

No irregularity to be made out in the cervical transverse processes. Urine sp. gr. 1024. No albumen, sugar, or blood.

28th.—Patient has great difficulty in swallowing this morning; but is otherwise the same. Evening temp. 103°.

29th.—Breathing quicker (resp. = 42), more laboured, râles more numerous. Abdomen much distended, intestinal coils visible. Surface of body covered with perspiration. Patient died at 10 p.m. Temperature before death = 103·8°.

Post-mortem.—On exposing the bodies of the cervical vertebræ there was found to be a gap between the fourth and fifth, the upper edge of the fifth being very prominent, but the amount of displacement very slight indeed, and in an unusual direction, for the body of the fourth vertebra was on a plane slightly posterior to that of the fifth, but not more than one eighth of an inch. The anterior common ligament was partly but not entirely ruptured, about half the fibres at the sides having given way. Posterior common ligament intact. No fracture of the spines or laminæ. On exposing the spinal cord there was practically no extra-meningeal hæmorrhage. The membranes between the fourth and sixth cervical vertebræ were bulging, and felt tense, as if distended, but on opening them the surface of the cord seemed quite healthy, and there was an absence of intra-meningeal hæmorrhage. When the cord was removed the portion that before felt tense, now felt more flaccid and less resistant than the rest of the cord. On section there was a marked central hæmorrhage occupying about an inch in the length of the cord, and limited in great part to the grey matter. At the centre of the hæmorrhagic portion there was extension into the surrounding white matter, but all around there was a circumferential zone of apparently healthy white matter. It was very curious to see how, in the extremities of the hæmorrhagic portion, the hæmorrhage was confined to the grey matter only.

On section of the vertebræ there was evidence of old caries in the bodies of the fourth and fifth cervical. These were extensively sclerosed, that of the fourth more so than that of the fifth. The inter-vertebral disc had disappeared, and there was now a cavity between these two bodies, produced by their forcible separation at the time of the accident. The arches of the third and fourth vertebræ had become ankylosed, so that there was probably no movement possible between these two vertebræ. It was especially noted that the amount of displacement of the upper vertebra was too slight to cause any compression of the cord. No atheroma of arteries found.

In this case, as in Case 1, there was complete paraplegia of motion and sensation corresponding to a lesion between the fifth and sixth cervical vertebræ. There were, however, distinct plantar reflexes and knee-jerks. Other points of interest are the curious reflex movements of the limbs, extreme myosis, and rise of temperature before death to 103·8°.

The situation of the abrasion on the forehead seemed to point to a hyper-extension of the head and neck as the cause of the injury to the spine, which was obviously weakened by the old caries between the fourth and fifth cervical bodies. The ankylosis of the third and fourth vertebræ above this point would cause any extension force to act with much greater advantage in the separation of the adjacent bodies of the two vertebræ immediately below.

Case 3. *Partial dislocation of cervical spine; intra-spinal hæmorrhage.*—Mary H—, æt. 40, admitted under Dr. Taylor, July 29th, 1889.

History of injury.—Patient was going home on July 27th, and, on reaching the passage leading from the front door, fell, striking the back of her neck against a step. She became unconscious and was carried into the house; her friends say that she has been unable to move at all since the accident. She was brought up to the hospital the following morning.

On admission.—Patient is conscious and able to give an account of the accident. Legs, abdomen, chest, and arms completely paralysed and flaccid; the right biceps could be, however, contracted very slightly. Anæsthesia complete below second rib in front of chest. No hyperæsthesia. Slight plantar reflex on each side. Knee-jerks, abdominal and epigastric reflexes absent. No ankle-clonus. Pupils contracted to a minute size, and scarcely react to light. Neither sterno-mastoids nor scaleni act in respiration, which is carried on only by the diaphragm. Patient is cyanosed and the chest full of râles. The line of the cervical transverse processes is distinctly altered on the left side. The fifth transverse process is prominent, in a line with those above but distinctly anterior to the sixth and seventh. No such deformity on the right side. Patient's head is turned to the right, and cannot be brought to the middle line. Pulse = 60. Resp. = 30.

At 8 p.m., four hours after admission, chloroform was given; extension of the spine did not reduce any of the deformity. The patient was, therefore, turned on her side, and an incision 4 inches long made vertically with its centre over the sixth spine by Mr. Lane. Spines and laminæ were exposed and those of the fifth and sixth vertebræ removed. Opposite the upper vertebra the cord was said to feel softened, and did not pulsate below this point. Membranes cut open, but the surface of the cord looked healthy. All attempts at reduction of the displacement failed, the vertebræ slipping back again immediately traction was discontinued. Wound closed, a drainage-tube inserted, and antiseptic dressings applied. The head was kept rigidly quiet by a casing made of plaster-of-Paris bandages. At the beginning of the operation pulse = 40, respiration very bad, legs blue. At the end of the operation pulse = 72, respiration easier, and cyanosis less marked.

July 29th.—Temp. 104°. Patient delirious, cyanosis marked. Plaster case cut open, wound dressed. Patient became more and more cyanosed and died at 1 p.m., about fifty-three hours after the accident.

Post-mortem.—On examining the spine in front, some undue mobility was found between the fifth and sixth cervical vertebræ, but there was no deformity to be noted and very little extravasation of blood. The anterior common ligament was partially torn. On further investigation it was seen that there was a partial dislocation between the two vertebræ, the inferior articular process of the upper vertebra on the left side having slipped in front of the superior articular process of the lower; the deformity was, therefore, rather a lateral rotation than an antero-posterior displacement. The intervertebral disc was torn off to a considerable extent, carrying with it tiny bits of bone which gave rise to the crepitus during life. A little blood had run down the canal, but not in sufficient quantity to do harm.

The cord looked quite healthy on the outside, but on section opposite the seat of injury it presented a mass which was described as something like strawberry cream; surrounding this was a rim of white matter which was not softened. Below this most damaged portion there was a continuation of the hæmorrhage, especially in the posterior root zone, extending into the upper dorsal region of the cord.

Under the microscope the red material in the cord showed much blood, droplets of myelin, and wrinkled, irregularly swollen axis cylinders.

This case resembles very much Cases 1 and 2, in that the symptoms pointed to a total transverse lesion.

The partial or one-sided dislocation of the cervical spine could scarcely produce a narrowing of the vertebral canal sufficient to compress the cord; and the inference appears to be that the injury was produced by a severe flexion of head and cervical spine accompanied by a blow upon the cervico-dorsal region, that portion of the neck coming in violent contact with the edge of the step against which the patient fell. The presumption is also that the patient fell somewhat sideways, *i. e.* towards the left.

The case resembles the previous ones in the loss of knee-jerks, the myosis, and hyperpyrexia.

CASE 4. *Fracture of 6th cervical vertebra (no displacement); intra-spinal hæmorrhage; compound fracture of right tibia; gangrene of right leg.*—J. A—, æt. 48, admitted January 5th, 1891, under the care of Mr. Howse.

History of accident.—Patient was moving a large ginger-beer vat, when the chain suspending it broke and the vat fell upon him. He was rendered unconscious, and cannot say where he was struck. He was brought immediately to the hospital.

On admission.—Patient was quite conscious but somewhat collapsed. There was a compound comminuted fracture of the upper end of the right tibia, with projection of the upper end of the lower fragment through the skin. Some effusion into the soft tissues around the fracture. The fibula was probably also broken at about the same level.

There was a scalp wound three quarters of an inch long in the median line of the head in the centre of the vertex leading down to bone.

The patient complained of loss of power in his arms and legs and of numbness in all his extremities, but at the time of admission, owing to the serious condition of the right leg, a minute examination was not made.

Treatment.—The scalp wound was dressed antiseptically, and the right leg put up in back and side splints and swung in a cradle.

January 6th.—Right leg was of a dusky colour, colder than left, with complete anæsthesia below the knee. Considerable effusion in the popliteal space, which felt quite hard, the swelling extending up the lower third of the thigh. No pulsation in arteries below knee. Patient could move left leg a little, but not his arms. He says they all feel numb, although he localises sensations very well all over the body. Good knee-jerk on left side. No ankle-clonus; when the sole is tickled he draws up left leg, but says he cannot do so of his own free will. No abdominal or epigastric reflexes. No thoracic or abdominal paralysis. Urine has to be drawn off by catheter. Sp. gr. 1025. No albumen, sugar, or blood.

Careful examination of the neck failed to reveal any irregularity in line of transverse processes or spines of the cervical vertebræ.

7th.—The temperature, which before had been subnormal, went up last night to 100·2°. No change in condition of right leg, but it was taken down from the cradle and allowed to remain flat so as to aid the arterial circulation. Other symptoms unaltered. There does not seem to be very good movement of diaphragm.

8th.—Patient complained of great pain in his arms, is still unable to move even his fingers. Left leg cannot be moved at all. In the centre of the left thigh and on the outer side are two blebs the size of four-shilling pieces; these have developed during the night. Right leg amputated just above the knee. On examining the limb removed there was found to be a laceration at the bifurcation of the popliteal artery, the two tibial arteries being almost torn away.

10th.—Diaphragm seems to be paralysed, epigastric region drawn in at each inspiration, chest moves well. Patient otherwise very much the same.

11th.—Urine strongly ammoniacal, sp. gr. 1016. Albumen present. No blood. Evening temp. 102·4°.

13th.—Left leg examined showed slight plantar reflex, good knee-jerk. No clonus. Hyperæsthesia in sole of left foot, the patient complaining of pain when touched here. Sensation otherwise seems good. Abdominal reflexes present, epigastric absent, cremasteric absent, complete loss of power in arms and legs. Patient does not look nearly so well. Evening temp.101·4°.

15th.—Scalp wound nearly healed. Stump appears to be healing satisfactorily. Urine, passed involuntarily, contains much pus, but no blood. In the morning patient became much worse; R.=30, P.=160, Temp. 100·6°.

16th.—Patient died at 8.40 a.m.

Post-mortem.—The laminæ of the sixth cervical vertebra were fractured; no fracture of the body or of the spine, no displacement. No extra-meningeal hæmorrhage. The cord corresponding to the seat of fracture was normal on section, and to naked eye there was a good distinction between grey and white matter. Higher up, about the level of the third cervical vertebra, the cord felt soft, and on section a localised hæmorrhage was found extending vertically for an inch, and apparently affecting only the central portion of the cord, the white matter around being normal and not at all soft. The hæmorrhagic part was of a brownish colour and almost diffluent; it corresponded to about the third vertebra in position and extent, though the latter was difficult to define. At the level of the hæmorrhage no injury to the bones could be made out.

Large arteries healthy. Kidneys 12 oz., slightly granular on the surface.

This case is decidedly peculiar from the irregular nature of the spinal symptoms. It was unfortunate that on admission a more complete examination was not made. The state of the left leg only could be made out on account of the serious nature of the injury to the right. There was paralysis of left leg and of both arms; no thoracic or abdominal paralysis. Two days after admission diaphragm said to be acting weakly, and later became paralysed. Sensation disturbed, paræsthesia, but localisation seemed to remain throughout. Left knee-jerk and plantar reflex present up to two days before death; other reflexes absent. No hyperpyrexia, no myosis.

The nature of the fracture suggests very strongly a direct blow, but no accurate history was obtainable.

CASE 5. *Injury to back; paraplegia; left brachioplegia; intra-spinal hæmorrhage; right Pott's fracture.*—W. S—, æt. 61, admitted March 29th, 1887, under the care of Mr. Durham.

History of accident.—Patient was standing underneath a lift, when the chain broke and the lift fell on to his head. He was immediately rendered unconscious, and remembers no more about the accident.

On admission.—Patient was quite conscious; had a scalp wound on the back of the head, and a Pott's fracture on right side. No signs of paralysis on carefully examining the limbs, but eight or ten hours after admission the left arm was found to be weak and rapidly became paralysed. No irregularity anywhere in spine.

March 30th.—Anæsthesia over both legs and arms, said to be only partial. He was able to move right arm a little, but not the left. Could not pass his urine; a catheter had to be used twice a day. Motions passed involuntarily. Temperature subnormal.

April 2nd.—Left arm completely paralysed. Left leg is held stiffly, and moves a little, though he says he cannot move it voluntarily. No knee-jerk on left side. Plantar and epigastric reflexes present. For the last twenty-four hours has been wandering in his mind, and this prevents any determination of the extent of anæsthesia; yesterday he said he could not feel at all in the left leg.

6th.—Complete left hemiplegia; anæsthesia about the same. The right leg is probably affected, though, owing to the fracture, this cannot be determined. Can raise right arm to the shoulder.

11th.—Condition much the same. Died quietly.

Post-mortem.—Spinal column intact. No extra-meningeal hæmorrhage. On making a section of the cord it appeared that there had been extensive hæmorrhage into, and destruction of, its central part at the level of the cervical enlargement. Above and below the actual lesion the cord to the naked eye appeared healthy. The most extensive mischief was at a point two and a half inches below the apex of the fourth ventricle.

Here the central part of the cord was hollowed out, having about one eighth of an inch of healthy periphery. The cavity contained soft grumous material, evidently altered blood, which escaped on section. The cavity occupied about three quarters of an inch of the length of the cord; it ended abruptly below, but above some discoloration and softening extended upwards in the central grey matter, reaching half an inch higher on the left side than on the right. Early broncho-pneumonia. Other viscera healthy.

This case differs from the previous ones in the gradual onset of the paralysis (eight or ten hours), resembling in this respect one reported by Dr. Sharkey in the 'Lancet,' May 23rd, 1891. Paralysis of left arm and probably of both legs became complete, knee-jerk on left side absent. Anæsthesia probably partial. There is no note of temperature or pupils.

The injury to the spinal cord was in all probability produced by hyperextension of the head and neck.

CASE 6. *Injury to back; complete paraplegia; extreme fall in temperature; intra-spinal hæmorrhage.*—G. P—, æt. 28, admitted December 9th, 1888, under the care of Mr. Howse.

History of injury.—Patient was going downstairs last night between eight and nine o'clock, with a heavy tool-box on his head, when he slipped upon the last step. The tool-box jarred heavily upon his spine, and he fell backwards. He is quite certain that his head did not come forcibly in contact with the stairs. He did not become unconscious, but was unable to move or even to call out. He was brought up to the hospital the morning after the accident at 7 o'clock.

On admission.—Patient quite conscious; complained of pain as if his "funny bone" were touched, locating this at the sixth cervical vertebra behind, but nowhere else. Nothing to be made out on careful examination of the spine. No movement of legs or chest, but he could move his left arm a little. Anæsthesia below a horizontal line drawn through the nipples; above this line there was a region of "hot prickly" feeling when he was touched. Priapism marked. Soon after admission forcible traction on the head and neck was made, under chloroform. Crepitus was said to be felt whilst the patient was under the anæsthetic (blood crepitus?). After the effects of the chloroform had passed off the symptoms remained the same. Pulse = 52. Resp. = 12. Temp. = 97·4°.

December 10th.—Patient has complete paraplegia up to and involving both arms; he can use the deltoids slightly. Breathing entirely diaphragmatic. Sensation good over deltoid muscles and above this level. Pupils a little dilated, but equal and react to light. Patellar jerk good on both sides. Slight plantar reflex on each side. Bladder distended; no desire to micturate.

12th.—A dusky red patch has appeared over each olecranon. Temp. 97°. Condition otherwise much the same. Urine acid, sp. gr. 1035. No albumen, sugar, or blood.

24th.—Bladder washed out daily with Thompson's fluid. There is a small sore, the size of a sixpenny piece, at the back of each elbow; each is surrounded by a slightly reddened area. Marked evidence of cystitis in the presence of blood and pus in the urine.

28th.—Patient now speaks feebly, and wanders considerably in his speech. The temperature has steadily fallen to 86°.[1]

29th.—The sores at the back of the elbows have healed, and no other bedsores have developed. Patient is only partly conscious; can recognise his friends. Much feebler, and declines his food.

January 2nd.—Temp. 78·2°. Resp.=15, shallow. Pulse cannot be counted. Patient has nystagmus and conjunctivitis. Pupils very contracted, and insensitive to light. Has had one motion since admission on December 23rd. Patient gradually sank, and died at midnight.

The gradual falls in temperature and pulse-rate are shown in the accompanying table.

	Temperature.			
	Morning.	Evening.	Pulse.	Respiration.
December 9th.	97·4°	99·8°	52	12
10th.	96·4	97	—	—
11th.	96	97·2	46	11
12th.	97	96·4	50	12
13th.	95·6	98	—	—
14th.	95·7	97·3	41	—
15th.	95·6	96	42	12
16th.	96·4	97·2	43	11
17th.	95·6	96·2	44	14
18th.	94·6	96	—	—
19th.	93·4	95	38	15
20th.	94	94	44	—
21st.	92	—	40	—
22nd.	92	—	—	—
23rd.	90·6	—	38	—
24th.	90·4	—	37	—
25th.	89	—	—	—
26th.	88	—	32	—
27th.	—	—	—	—
28th.	86	—	34	11
29th.	85	—	—	—
30th.	82	—	—	—
31st.	81·4	79·8	26	18
January 1st.	79	78·4	Not countable	19
2nd.	78	77·6	,,	15

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[1] Every possible care was taken by Dr. Samways, who reported the case, that the temperatures should be accurately recorded. A special thermometer was used.

Post-mortem.—There was no displacement of any of the vertebræ, but the bodies of the fifth and sixth cervical were easily separable on account of the destruction of the intervertebral disc; there was a small rough surface on the body of the fifth, as though a small scale of bone had been chipped off. No fracture of the laminæ or spines.

Meninges of cord were normal, but the cord itself on section showed a diffluent mass for one and a half inches of its length, extending from about the fifth to the seventh vertebra; the material which oozed out on section was like dirty cream. The upper part of the pulped cord was deeply blood-stained, and through the comparatively healthy cord above, there extended a narrow channel containing "purulent" material as high as the calamus scriptorius. No naked-eye change of descending degeneration.

There was nothing in the vertebræ found post mortem to indicate that a dislocation had at any time been present; it was *possible* that a dislocation might have existed at first, and that it had been reduced by traction, but from the condition of the parts it seemed very unlikely in the opinion of those who saw the spine after death. The case was regarded as one of "local concussion, or laceration of the cord going on to softening or destruction; the spinal canal at the seat of injury being unencroached upon" (*vide* 'Guy's Hospital Gazette,' March 2nd, 1889, vol. iii).

A number of superficial ulcers filled with adherent blood-clot were found along the posterior curvature of the stomach towards the pyloric end. A few small patches of broncho-pneumonia.

This case resembles the first three in that the symptoms point to a total transverse lesion, and here again the knee-jerks were absent. The pupils, at first dilated, became later much contracted. A curious feature was the fall in temperature and pulse-rate.

It seems possible that there was a direct blow from the edge of the box upon the lower cervical spine, as a mere jarring of the box would probably have produced a fracture dislocation in the upper dorsal region.

From the early onset of the symptoms this appears to be an undoubted case of intra-spinal hæmorrhage; the "cream-like" material that oozed from the cord was obviously blood and broken-down nerve matter that had been undergoing various changes for the twenty-four days during which the patient lived. It appears unnecessary to imagine that the hæmorrhage was complicated by a central myelitis.

Case 7. *Injury to the cervico-dorsal region ; brachioplegia ; spastic paraplegia ; intra-spinal hæmorrhage ? ; intra-meningeal hæmorrhage ?.*—W. F—, æt. 25, admitted December 31st, 1886, under care of Mr. Durham ; discharged August 29th, 1887.

History of injury.—Patient was carrying a case of oranges weighing 2½ cwt. on his back, when he slipped on a plank and fell. It appeared that he sat down on the plank, and that the case rebounded on his shoulders, causing his head to be forcibly flexed on to the sternum. He was rendered insensible for a few minutes.

On admission.—Patient was somewhat collapsed. Examination showed that the left leg was weaker than the right, and there appeared to be irregular loss of sensation. Great weakness in both forearms, with loss of power of extension. Irregular anæsthesia in arms, with pain of a neuralgic character. Reflexes said to be normal. Retention of urine. Chest expands very slightly.

January 3rd.—Pupils equal, contracted. Pain in upper dorsal region of back, shooting down arms. A band of hyperæsthesia about five inches wide extending downwards from the fifth rib.

7th.—No improvement in power of arms. Can still flex the arms fairly well, but cannot extend the fingers or grasp with the hand. Urine passed reflexly when bladder is full, without any knowledge on the part of the patient.

11th.—A condition of spasm of the lower limbs has developed ; the legs become rigid occasionally, and then are flexed or extended forcibly. No ankle-clonus. No plantar reflex. Condition of arms remains same ; power of flexion of fingers, forearm and arm good, but that of extension seems quite absent. Pronation and supination weak. Some pain on rotation of neck.

14th.—Pain in arms more severe. Has pain in his back, which is easier when he rolls on to his side. Can tell which leg is touched, but does not locate well. Breathes with his diaphragm only. Cannot move his feet.

15th.—Muscles of upper and lower extremities respond readily to galvanism. No polar changes. Can only just feel a strong current in lower limbs, but exclaims with pain when same current is applied to upper limbs.

22nd.—Patient seems worse, cannot move arms at all, spasms in leg are more marked.

February 3rd.—Can extend arms a little, and can move left thumb.

March 1st.—Paralysis of extensors of arms is very complete. Slight power of flexion of right fingers, which are kept bent into the palm. No power of extension of right fingers or wrist. Cannot oppose the thumb. Left wrist moves well, the three outer fingers are flexed into the palm at the first phalangeal joint. Can oppose the thumb. No definite anæsthesia, but he says the left hand feels numb. When the legs are stretched out there is a clonic action of flexors of thigh.

5th.—Knee-jerks exaggerated, ankle-clonus well marked. Pain in legs still present, chiefly following course of obturator nerves.

July 12th.—*Right hand* "en griffe" with hardly any power of extension. Muscles of hypothenar eminence much wasted. Sensation fairly good. No trophic lesions. Thumb cannot be opposed.

Right forearm.—Wasting of flexor carpi ulnaris. Flexion of wrist slow but complete. Pronation slow and weak. Supination very good. Sensation a little impaired. A few scattered patches of hyperæmia. Extension of right forearm not very strong; outer head of biceps wasted.

Left hand shows rigidity of fingers, with abduction of little finger and flexion of the two middle fingers. They are not fixed in this position, but are capable of full passive movement. Thumb can be opposed. No numbness or anæsthesia. Other parts of left upper extremity seem normal. Ankle-clonus and knee-jerks very active; still has convulsive jerks of the leg muscles. Feet cannot be got to a right angle with legs on account of rigidity of calf muscles. Has abnormal sensations in legs at times.

August 29th.—Discharged, condition as above.

The interest of this case lies mainly in the severity of the symptoms, and the ultimate convalescence with persistency of spastic paraplegia. The case has been included under the idea that it was possibly one of localised intra-spinal hæmorrhage at the origins of the sixth and seventh cervical nerves, that is, about opposite the fifth and sixth cervical vertebræ, this hæmorrhage being in the grey matter extending into the lateral columns, and partly destroying the posterior columns.

It is perhaps more probable that the case was one of intra-meningeal hæmorrhage on account of the marked extrinsic symptoms—neuralgic pains, pain over the spine, &c., followed by painful spasm in the lower extremities, due possibly to extension downwards of the hæmorrhage.

It is extremely difficult to say whether the spastic condition which developed later ought to be ascribed to a chronic myelitis following a meningeal hæmorrhage, or to changes in the lateral columns the result of intra-spinal hæmorrhage. There appears to be no doubt that there was a want of evidence at the time of any lesion to the vertebral column itself, the case being regarded as one variety of spinal concussion.

Microscopical examination of the cord was made in Cases 1, 2, and 4. In each case the centre of the section showed a mass of red corpuscles which occupied the place of the grey matter, and which was surrounded by the nerve-fibres of the white matter. No tracts of degeneration could be seen. Around the blood-clot in Case 4 there seemed to be an excess of leucocytes, as if changes in the clot were already advancing. No "mulberry" masses found in any of the sections.

Remarks.

These cases illustrate very well the symptoms produced by intra-spinal hæmorrhage. As the various points of interest in each instance have already been alluded to, it only remains to consider a few details common to all. The last case, being of a doubtful nature, will not receive further attention.

All the cases were the result of injury, so that it is not necessary to do more than allude to other alleged causes of hæmato-myelia, such as congestion of the cord, sudden alteration in blood-pressure, severe bodily exertion, and others, many of which are more problematical than exact. That injury is the common cause admits of no question, and in all probability the so-called cases of severe spinal concussion are instances of the affection under consideration. The exact way in which hæmorrhage occurs is not very obvious. Many contend that the cord is so loosely attached to the vertebræ, and is so well protected by its membranes and subarachnoid fluid, that any injury to it alone is almost an impossibility. That this view is not correct is evidenced by Case 5.

In all the cases the hæmorrhage was situated in the cervical region, and, with the exception of Case 4, in the most prominent part of the cervical enlargement, opposite to the bodies of the fifth and sixth vertebræ; here the transverse diameter of the cord is greatest, and there is least space between it and the sides of the vertebral canal. Perhaps considerations of greater value are, firstly, that at the root of the neck is the junction of the least mobile with the most rigid part of the vertebral column; and secondly, that of the movements of the cervical spine extension is permitted to a greater extent than flexion. It is consequently only fair to suppose that in the cervico-dorsal region (probably at the junction of the sixth and seventh cervical vertebræ) there is the greatest liability to displacement of the vertebræ, and the greatest danger to the cord in cases where excessive movement takes place.

A similar line of argument accounts for the fact that the dorso-lumbar region is the part of the spine that, next to the lower cervical region, is most frequently fractured.

The exact way in which the various injuries were produced in the above cases cannot be made out with absolute certainty. The number of cases is also too small to draw any exact conclusions. In Cases 1, 3, and 4 it seemed very probable that the spinal hæmorrhage was the result of direct violence; in Cases 2 and 5 there was apparently hyperextension of the spine; while in Case 3 unilateral flexion of the spine seemed to accompany direct violence. In Case 6 the exact nature of the accident could not be made out. It seems fair to conclude for the present that either a localised blow on the spine or hyperextension in the cervical region may produce hæmato-myelia. A fracture dislocation of the spine is, on the other hand, generally associated with excessive flexion.

In all the cases it was noticed that the hæmorrhage was central, and it appears to be the general rule that although hæmorrhage may by extension destroy the white matter, it never primarily arises in that portion of the cord; and as it extends up or down the cord, it does so along the course of the grey columns. When such a cord is exposed post mortem the tense bulging of the affected part can be readily perceived by the finger; it is only when the cord has been removed from the canal that the diseased part feels softer than the healthy portion. There seems to be no reason why a hæmorrhage into the cord should not be as easily recognised during life, if the laminæ are removed, as it is after death; but whether an incision into the damaged part along the posterior median fissure to relieve tension would be beneficial, or not, can only be speculated upon. Considering the fatal nature of the injury it would be advisable to try.

The symptoms in the cases given above vary greatly. In Cases 1, 2, 3, 6, there was complete loss of motion and sensation up to about the same level, and in Cases 1 and 3 every evidence of a total transverse lesion. In Case 2 the knee-jerks remained, and this ought to have suggested the nature of the case. In Case 4 there was complete paralysis of arms; paralysis of legs—partial at first, with slight disturbance of sensation only; knee-jerks present, and breathing entirely by thoracic muscles—a condition clearly indicating a small lesion opposite the third and fourth cervical vertebræ, *i. e.* a local lesion destroying the centres for the diaphragm and arms, but not interfering considerably with the lateral tracts on either side. Case 5 resembles very much Case 4, and these two illustrate very well the fact that immediate paralysis of both arms following an injury, with only slight loss of power below, and little alteration in sensation, can only be due to an intra-spinal hæmorrhage.

It is obvious that the symptoms in any case of hæmatomyelia depend rather upon the transverse than upon the vertical extent of the lesion. A small lesion confined to the grey matter will be evidenced only by paralysis of the muscles whose centres are in the anterior cornual cells of the affected part, with a varying but slight alteration in sensation of approximately corresponding parts; whilst a larger lesion will produce much more marked effects by irritation or destruction of the columns of white matter; so that the symptoms may vary from a local paralysis with slight sensory disturbance, to a total paralysis and complete loss of sensation indicative of a total transverse lesion or section of the cord.

Diagnosis.

Intra-spinal hæmorrhage has to be distinguished from the following conditions. (1) Fracture-dislocation of the spine with crushed cord. (2) Myelitis centralis hæmorrhagica. (3) Meningeal hæmorrhage (hæmatorrhachis). It need scarcely be said that the differential diagnosis is in many cases extremely difficult.

1. In fracture-dislocation of the cervical region there are several points which render valuable assistance in the diagnosis.

A vertical force acting upon an acutely flexed spine is the common cause, of which many instances could be given. A curious one is a mode of assault at one time very frequent in Lancashire. One man would seize another from behind, passing his arms underneath his opponent's axillæ, and clasping them behind his neck; an amount of power could then be exerted upon the cervical spine which would readily produce a fracture-dislocation.

Irregularity in the cervical spinous processes is not easily made out except between the sixth and seventh. Local tenderness over the spines is not much guide, being often the result only of a severe sprain.

Very good evidence of a dislocation is obtained by tracing down on each side of the neck the line of the transverse processes. If there is a break in this line, the two parts of which tend to overlap obliquely, there is no doubt of the displacement of the bodies; this could readily be made out in Case 3.

Severe pain on passive rotation of the neck, with loss of power of active rotation, helps in the diagnosis of fracture-dislocation. Crepitus, especially under an anæsthetic, needs scarcely to be mentioned. In those cases of spinal hæmorrhage that I have seen, there was a marked absence of any ring of hyperæsthesia. Paræsthesia in different places was common.

It is of course obvious that there is no reason for any ring of hyperæsthesia, or, what corresponds to it in the cervical region, neuralgic pain down the arms. In cases of fracture-dislocation what happens is that the inferior articular processes of the upper vertebra, on account of the flexion glide upwards over the superior processes of the vertebra below; this movement is carried to an excess, and they suddenly shoot forward in front of the latter, so that severe compression of the nerve-roots is produced by their being nipped between the displaced inferior processes above and the laminæ below. The deformity thus produced is most difficult to remedy. I have tried direct traction post mortem on two cases with but little result, although the front of the spine was bare and any movement could be easily directed. Reduction could be effected only by first flexing the head and neck forcibly, then making traction to tilt the lower processes of the upper vertebra over the upper processes of the lower, followed by extension. It is to my mind very questionable whether such manipulation on the living subject would not be likely to injure the cord very severely, and whether the best result would not be produced by laminectomy. Even when laminectomy is done the obstacle to reduction may still be overlooked; and, unless the superior articular processes of the lower vertebra are carefully removed, it is very doubtful if reduction could be brought about, unless, say, at the time of the accident both processes had been broken off. This mode of interlocking is common to fracture-dislocation in all parts of the spinal column.

In Case 3 the diagnosis of unilateral dislocation was obvious from the deformity of the transverse processes existing on one side only and from the head being rotated towards the opposite side, and fixed in that position.

2. *Hæmorrhagic myelitis* has frequently been confused with hæmatomyelia, especially in those cases where the myelitis extended rapidly along the length of the cord. It might, indeed, be argued that Case 6 was one of this kind, but the onset of paraplegia was so definitely sudden, and apparently without displacement of the vertebræ, that no other diagnosis but that of primary hæmorrhage into the cord would be satisfactory. Whether the hæmorrhage subsequently extended upwards, or whether a central myelitis ensued, cannot be decided in the absence of all microscopical evidence. It may be shortly stated that hæmorrhagic myelitis takes at least some hours to develop; it begins with symptoms of irritation, pain, slight spasm, paræsthesia or anæsthesia, with tenderness over the vertebræ. Partial paralysis of the bladder precedes very frequently the onset of severe paraplegia, and the general temperature is raised.

On the other hand, as Ziemssen states, the characteristic features of extensive hæmatomyelia are sudden and very rapid invasion of paraplegia usually attended by a violent pain at the onset, localised or extending over the spinal column, but disappearing after the palsy has become complete.

3. *Spinal meningeal hæmorrhage* (hæmatorrhachis) is characterised by the sudden onset of symptoms which denote meningeal irritation (extrinsic symptoms due to pressure upon the nerve-roots) *e. g.* violent pain radiating along the course of the nerves affected, hyperæsthesia and paræsthesia with spasmodic jerkings of the muscles, which, according to Brown-Séquard, never occur in hæmatomyelia. The condition of the muscles below the lesion varies from that of weakness to that of complete paralysis; if the hæmorrhage is in the cervical region the paralysis of the upper extremity is greater than that of the lower. To sum up, the symptoms are due to irritation of the nerve-roots and of the cord, followed by compression of the cord. In this class of cases the prognosis is better than in cases of intra-spinal hæmorrhage.

Reflexes.

The condition of the reflexes in cases of damage to the spinal cord is one that is of interest from the statement of Dr. H. Charlton Bastian, in the 'Medico-Chirurgical Transactions,' vol. lxxiii, to the effect that when there is a complete section of the cord all the reflexes below the point of section disappear; and that rigidity of the lower limbs with increased reflexes indicates that the section of the cord is incomplete. This view received support from Mr. Anthony Bowlby, who quoted a number of cases of injury to the spine, in which the condition of the reflexes had been carefully observed. The two adjoining tables contain all the cases of fracture-dislocation of the spine and of intra-spinal hæmorrhage which have been admitted into Guy's during the last eight years, those cases only being included, in the reports of which a definite note of the reflexes and jerks was made, and confirmed by the Surgical Registrar for the time being. It is to be regretted that in many other cases no mention is made of the reflexes, so that a large amount of material is for this purpose of no use.

If these tables be considered, it will be seen that the cases in the main support the view above stated, but exception must be taken in Table I to Cases 8 and 10, and in Table II to Case 2.

In the two former cases there was a permanent condition of complete paraplegia and anæsthesia with marked rigidity of the limbs, and in each a slight contraction of the quadriceps on striking the ligamentum patellæ put down as ? knee-jerks, ? idio-muscular contraction.

If we take with Dr. Bastian complete paraplegia and anæsthesia below a certain level as diagnostic of a total transverse lesion, we have here two such cases with rigidity of the limbs and knee-jerks. If, on the other hand, the lesions were not total, we have two cases of a partial lesion, in which the knee-jerks were not exaggerated, to say the least; in which there was no ankle-clonus, and an unusual feature—complete anæsthesia. These two cases, therefore, lend strong support to the older view that when there is a transverse section of the cord, the deep reflexes become exaggerated and muscular rigidity develops. Both these cases have been under my own observation.

A third case which does not seem to substantiate Dr. Bastian's view is No. 2, Table II; but as this is a case of intra-spinal hæmorrhage it is impossible to be certain that the lesion was completely transverse; in fact, the curious unconscious movements of the legs support the opinion that a portion of the lateral tract remained.

The question may be raised whether the plantar reflex and knee-jerk do not become altered in different ways under the same circumstances; this appears to be so from the adjoining tables, but from them alone I do not feel capable of forming any definite opinion, either on this point or upon the major question. I feel sure that there remain many points about deep reflexes upon which further investigation will throw much light.

A difficulty that often arises is that of recognising whether a contraction of the quadriceps extensor muscle, which produces no movement of the leg, is a very slight knee-jerk or an idio-muscular contraction; it can certainly be produced by a blow upon the muscle as well as by a blow upon the tendon, but this, in the absence of exact time measurements, does not seem to be decisive. Dr. Bastian says that in the case of a progressive cord lesion this idio-muscular activity is the last remnant of motor power in the muscles, but in the case in question when does the knee-jerk disappear, and the idio-muscular activity alone remain?

Again, a further difficulty arises when the muscles are so rigidly contracted that not the slightest movement of the limb is permitted, and consequently no ankle-clonus or knee-jerk possible. In these cases on trying to obtain a knee-jerk a slight contraction in the quadriceps can often be felt by the hand on the muscle.

That the disappearance of all reflexes after a crush of the cord may be due to shock has been frequently stated. Whether such shock could last several days even, is very doubtful, but it also seems unlikely that with a crushed cord, incapable of transmitting sensory impressions, much or any effect on the sensorium could be produced. Patients

TABLE I.—*State of Reflexes in Cases of Fracture-dislocation of Spine with crushed cord.*

	Position of lesion.	Reflexes on admission.	Reflexes at a later period.	Duration of case.	Remarks.
I	Cervical 5th–6th	All reflexes absent	—	Died 1 day after admission	Anæsthesia complete.
II	,, 6th	,,	—	Died 1 day after admission	,,
III	,, 6th	,,	All absent (3rd day)	Died 3 days after admission	,,
IV	,, 6th–7th	,,	? All absent (4th day)	Died 4 days after admission	Partial anæsthesia.
V	,, 7th, dorsal 1st	,,	Plantar present (7 mos.); K. ? (idio-muscular contraction ?)	Discharged 7 months after admission	Slight return of sensation.
VI	Cervical 7th, dorsal 1st	,,	All absent (2nd day)	Died 2 days after admission	—
VII	Dorsal 6th–7th	Pl., Abd., present; Cr. exaggerated; Ac. absent	Pl., present; Abd., K., absent; Ac. absent (6 mos.)	Discharged 6 months after admission	No rigidity of limbs; anæsthesia below 6th rib.
VIII	,, 5th–6th	Pl., present; Ac., Abd., Cr., absent	Pl., Abd., Ep., Ac., absent; K. ? (idio-muscular contraction ?)	Still under observation	Rigidity of limbs; permanent anæsthesia below 6th rib.
IX	,, 10th–11th	Pl., K., Ac., absent; L. Cr. absent; R. Cr. present	Pl., Abd., Ep., present; K. exaggerated; Ac. on L. side (3 months)	Still under observation, 3 months	Complete return of sensation; slight return of movement.
X	Dorso-lumbar	K., Pl., Ac., absent	K. absent ? (idio-muscular contraction ?); Pl. exaggerated; Ac. on R. side (8 months)	Discharged 8 months after admission	Anæsthesia below level of lesion; rigidity of limbs.

Pl. = Plantar reflex.
Abd. = Abdominal reflex.
K. = Knee-jerk.
Cr. = Cremasteric reflex.
Ep. = Epigastric reflex.
Ac. = Ankle-clonus.

TABLE II.—*State of Reflexes in Cases of Intra-spinal Hæmorrhage.*

	Position of lesion.	Reflexes on admission.	Reflexes at a later period.	Duration of case.	Remarks.
I	Cervical 6th	L. Pl. present? R. Pl., K., Ac., Cr., Abd., absent	All absent up to death	2 days	Complete anæsthesia below 2nd rib.
II	„ 4th–5th	Pl. marked; no Ac.; idio-muscular contraction of quadriceps; Abd., Ep., Cr., absent	Pl. and K. good on both sides; no Ac.	3 days	Marked reflex movements of legs; anæsthesia below 2nd rib.
III	„ 5th–6th	Pl. slight; K., Abd., Ep., Ac., absent	—	2 days	Anæsthesia below 2nd rib.
IV	„ 6th	Pl. and K. good; Abd., Ep., absent	Pl. and K. good (9 days); Abd. present; Ep. absent	11 days	No anæsthesia; paræsthesia.
V	„ 7th (about)	Not stated	K. absent; Pl., Ep., present (4 days)	13 days	Partial anæsthesia.
VI	„ 5th–6th	Pl. slight; K. good	Not given	24 days	Complete anæsthesia below 3rd rib.

Pl. = Plantar reflex. Abd. = Abdominal reflex. K. = Knee-jerk.
Cr. = Cremasteric reflex. Ep. = Epigastric reflex. Ac. = Ankle-clonus.

admitted immediately after an accident, causing complete section of the cord, rarely show much evidence of general collapse; the pulse is usually regular and little quickened, and there is not often much pallor. Consciousness, if lost at all, usually returns a few minutes after the accident.

I have only come across one case which at all supports this idea. It was put down as a case of spinal concussion; the knee-jerks at first were lost but returned in two days, and after two months became exaggerated, with other evidences of spastic paraplegia.

Hyperpyrexia.

In cases of intra-spinal hæmorrhage, as in many cases of fracture-dislocation with crushed cord, it is not unusual for death to be preceded by hyperpyrexia. This was marked in cases 1, 2, and 3, in which the temperature before death rose to 107·6°, 103·8°, and 104° respectively, and temperatures higher than these are by no means uncommon; in fact in some cases a temperature of 110° has been recorded after death. This hyperpyrexia is found accompanying lesions between the fifth and seventh cervical vertebræ, but is by no means a constant feature after damage to the cervical portion of the cord. In Case 4 the lesion was opposite the fourth cervical, and no hyperpyrexia resulted. In Case 6 there was a curious gradual fall in temperature to 78·2°; this fall in temperature has been noted in some cervical cord affections, and may in this case be possibly associated with the extension of the mischief up along the grey matter nearly to the fourth ventricle.

The explanation of these phenomena is difficult. It has been shown by Dr. Hale White and others that there is in all probability a heat centre in the lower part of the cervical cord; and as damage between the fifth and seventh vertebræ is accompanied by hyperpyrexia, it seems possible that in this region there is a centre which controls thermogenesis, especially in the splanchnic area. If this centre be destroyed an excess of heat will be produced, which in the absence of a corresponding loss will tend to raise the general body temperature. Hyperpyrexia in the cases under consideration does not come on for twenty-four hours after the accident, and is often accompanied by profuse sweating.

The fall in temperature in other cases may be explained by supposing that the thermal centre remains intact, actively controlling thermogenesis; all connection with the higher cerebral centres being cut off by a lesion above it, thermolysis is in excess of thermogenesis. In Case 4 it is necessary to suppose that the tracts connecting this thermal centre with the upper part of the brain remained intact, the lesion being only small. It is obvious that these explanations are purely speculative, but in the absence of more data it is impossible to emerge from this speculative region.

Myosis.

The extreme myosis noted in Cases 1, 2, 3, and 6 is a common result of injury to the cervical spinal cord opposite the fifth to the seventh vertebræ. As a symptom in such cases it appears to be a constant feature, and is usually ascribed to destruction of the cilio-spinal (pupil-dilating) centre situated in this region. Like hyperpyrexia the myosis comes on about twelve to twenty-four hours after the accident.

Prognosis.

Little can be said about the prognosis of cases of hæmatomyelia. The diagnosis is often obscure and seldom certain until after a post-mortem, except perhaps after operation. In cases that recover (necessarily slight) the diagnosis is always doubtful.

Ziemssen says, "Partial recovery often occurs, and cicatrization of the lesion in the cord takes place, but the recovery is generally incomplete." This appears to me to be a too sanguine view of cases which, if of appreciable extent in the cervical region, are almost of necessity fatal. Hæmorrhages lower down in the cord have a less grave prognosis, but their occurrence is of limited frequency.

There is no doubt that death is often brought about in cervical lesions by paralysis of the abdominal muscles and of the splanchnic nerves allowing the intestines to become overdistended, and so impeding the action of the diaphragm, which muscle is aided only by the sterno-mastoids and scaleni.

In the cases I have seen death has been preceded by profuse universal sweating accompanying cyanosis, myosis, and hyperpyrexia.

AN EXAMINATION OF FIFTY CASES

OF

MALIGNANT DISEASE OF THE STOMACH.

By E. C. PERRY, M.D., and L. E. SHAW, M.D.

Whilst engaged upon the preparation of a new edition of the Catalogue of the Pathological Museum at Guy's Hospital, we have lately had to examine about fifty specimens of malignant growths in the stomach, all of which were to be found upon our shelves, the earliest having been placed there in the year 1826, whilst the latest was added in the course of the last twelve months. Such a collection of morbid growths appeared to us to afford a substanial basis for an enquiry into the macroscopical and microscopical characters of gastric neoplasms, and we have added thereto such further results as we were able to deduce from the clinical accounts preserved in forty out of the forty-eight cases tabulated in this paper.

"The stomach," says Rindfleisch, "has an especially rich *repertoire* of carcinomata; we find with nearly equal frequency a soft, a hard, and a colloid form of glandular cancer, as well as a columnar epithelioma." Our own classification divides the gastric carcinomata primarily into cylindrical and spheroidal carcinomata. In either division the fibrous stroma may be abundant, in which case they are scirrhous cylindrical, or scirrhous spheroidal carcinomata, or it may be scanty, when they are medullary cylindrical or spheroidal carcinomata; or, finally, the epithelioid cells may have undergone colloid change, in which case we call them cylindrical or spheroidal carcinomata with much or little colloid degeneration.

Of cylindrical carcinomata we have twelve specimens (Cases 1 to 12), six of which were removed from the bodies of males, and four from females; in the remainder the sex is uncertain. Of spheroidal carcinomata we have thirty-two specimens (Cases 13 to 44), of which fifteen are males, and eleven are females, whilst of the rest the sex is doubtful. As our specimens were mounted for the most part with no knowledge of their histological character, it is probable that the above numbers represent with tolerable accuracy the relative frequency of cylindrical and spheroidal carcinoma. If this be true, Dr. Fagge has understated the frequency of cylindrical carcinoma when he says ('Principles and Practice of Medicine,' ed. 1, vol. ii, p. 149), "In a very few cases a cancerous growth in the stomach has presented the characters of a cylinder epithelioma."

A few words are necessary with reference to the histological character of the growths termed by us cylindrical carcinomata. By other writers similar growths are called columnar epitheliomata or carcinomata, cylinder epitheliomata, adeno-carcinomata, or destructive adenomata. As the normal epithelium of the stomach is indifferently described as composed of cylindrical or columnar cells, it appears to be of little importance which term we employ, more especially since, in many cases, the epithelium lining the tubes or alveoli of the growths, strictly speaking, is neither cylindrical nor columnar, but rather short columnar or cubical, resembling in fact that which is found in the glands of the stomach. A still further distinction is made by Ziegler in his work on 'Pathological Anatomy,' between a destructive adenoma, and a cylindrical epithelial carcinoma. As regards the former growth, the name adenoma is justified by the presence of a well-formed cylindrical epithelium lining in single layer the glandular tubules, which are themselves of large size, and exhibit a clear lumen. In the cylindrical epithelial carcinoma, on the other hand, the "glands are distended into more or less globular nests" filled by epithelioid cells, which, "as the result of mutual compression, assume very various forms, and retain their columnar character only at the periphery."

Accepting this subdivision of cylindrical carcinomata, we find that of destructive adenomata we have three characteristic examples (Cases 1, 2, and 3); but even in these it is possible to observe in parts of the sections that some of the tubules are filled with granular material and others contain clusters of spheroidal cells, and thus a transition is easily effected to the cylindrical epithelial carcinomata of Ziegler. Similarly, in the case of nine specimens (Cases 4 to 12), which fall under the type of cylindrical epithelial carcinomata, we have seen here and there tubules or "globular nests" so closely packed with spheroidal cells as to render it difficult, if not impossible, to demonstrate the existence of a peripheral lining of cylindrical epithelium.

In the spheroidal carcinomata the alveoli vary much in size, whilst the epithelioid cells are often much altered in shape. In some examples of diffuse infiltrating carcinoma with much stroma—india-rubber bottle stomach—these cells are arranged in rows as in scirrhous carcinoma of the breast, and are irregular and deformed apparently by the pressure of the fibrous tissue around them. To the appearances presented by this contracting form of cancer we shall return at a later period in the paper.

The fibrous stroma of the destructive adenomata is scanty, and in the cylindrical group generally the stroma is less abundant on the average than in the spheroidal carcinomata. Hence arises the greater tendency to contraction which characterises the spheroidal group, and is in some cases so great as to result in the production of the "india-rubber bottle" stomach mentioned above.

Colloid degeneration appears to be much more common in spheroidal than in cylindrical carcinomata. We have observed it in one out of twelve cases of cylindrical, and in nine out of thirty-two cases of spheroidal carcinomata. In nearly all these cases it was easily detected without microscopical examination by the gelatinous appearance of the growth. In Case 11 we have stated that "the edges of the growth are well defined, its surface is ulcerated, and has a gelatinous appearance somewhat resembling boiled sago-grains." In Case 36 the base of the malignant ulcer "presents a reticulated appearance, which is due to the falling out of the colloid granules," and in Case 31 there was "pitting" of the surface due to a similar cause.

Having thus investigated the microscopical characters of the growths and classified them accordingly, we proceeded to consider whether the various groups had any macroscopical characteristics which might serve for differentiation in the absence of microscopical examination; whether they had any special predilections as regards situation; and whether, finally, any help was to be gained from a consideration of age, sex, and frequency or situation of secondary deposits, where such were found.

Macroscopically, malignant growths may be distinguished by their position; by the condition of the mucous membrane covering them, which may be normally rugose, smooth, stretched, villous, gelatinous, or ulcerated; and by their shape and extent, whether they are raised and projecting internally or externally, or appearing as an infiltration of the gastric wall, either circumscribed or diffuse, with or without contraction. Moreover, a section through the growth will show to what extent the various coats of the viscus are involved.

Except in the case of the villous growths mentioned below, the mucous membrane gives no indication as to the histology of the growth. Colloid degeneration is generally readily recognised by the appearances above described.

Circumscribed globular or subglobular tumours are more commonly cylindrical than spheroidal carcinomata. On the other hand, the diffuse infiltrating growth, especially when accompanied with contraction, is in our experience invariably spheroidal. In the single specimen of an infiltrating diffuse carcinoma, which histologically has the characters of a cylindrical carcinoma, the stomach is little if at all less than its normal size.

As regards infiltration of the coats of the stomach it remains true for both classes of cancer that the greater part of the deposit is in the submucosa, and that the medullary cancers invade and destroy the muscular and serous coats, and project externally much more freely than the scirrhous growths, whether cylindrical or spheroidal. It is also true that on the whole medullary cancer is more common at the cardiac than at the pyloric end.

Two of our specimens, Cases 2 and 7, are at once distinguished from the rest by the villous condition of the mucous membrane covering the growth. In Case 7 the villi are slender processes varying from a quarter of an inch to an inch in length ; in the other case they are shorter, and some of them have small knobs at their ends, so that they might be described as small polypi with long pedicles. Histologically, the latter growth is a destructive adenoma, whilst the former is a cylindrical epithelial carcinoma in Ziegler's classification. The position of these growths is shown on the accompanying plates, and we have compared them in this respect with the two examples of non-malignant villous tumours of the stomach which are on our shelves (Cases 49 and 50). It will be seen that as regards locality the villous growths have no special predilections. In both of the malignant cases there was considerable ulceration, extending in Case 7 to the adherent liver, which presented a large cavity, the walls of which were formed of secondary deposit, with a villous surface resembling that of the primary growth. There were also numerous secondary deposits elsewhere. Of Case 2 we have no clinical account. As villous growths, *e.g.* of the bladder, are apt to bleed profusely, it is perhaps interesting to note that in Case 7 there was only slight hæmatemesis, and that the two non-malignant villous tumours did not cause any hæmorrhage at all.

With regard to locality, we have thought it convenient to give a diagramatic representation of the part of the stomach wall affected in each case of cylindrical and spheroidal carcinoma. (See plates.) The results may be summarised as follows:

Cylindrical Carcinomata (including the destructive adenomata and cylindrical epithelial carcinomata of Ziegler). Eleven cases available : 5 limited to the pylorus and encircling it (Cases 1—5) ; 1 involving the pylorus, lesser curvature and cardia (Case 9) ; 2 involving the lesser curvature and cardia

(Cases 7, 8); 1 involving the entire organ (Case 10); 1 involving the pyloric two thirds of the organ (Case 6); 1 localised on the posterior wall (Case 11).

Spheroidal Carcinomata. Thirty cases available: 11 cases limited to the pylorus (non-encircling 3, encircling 8), (Cases 13—23); 1 involving pylorus, lesser curvature and cardia, (Case 36); 2 involving the lesser curvature and cardia (Cases 37, 38); 7 involving the entire organ (Cases 29–35); 1 involving the middle third and encircling it (Case 28); 2 involving the lesser curvature alone (Cases 26, 27); 2 involving the lesser curvature and pylorus (Cases 24, 25); 3 limited to the œsophageal opening (Cases 40, 41, 42); 1 limited to the cardia and fundus (Case 39).

From this summary it appears that the pylorus is involved in 70 per. cent of the cases, the frequency with which it is involved being the same in both classes. There is only one specimen in which the fundus is involved except in the diffuse infiltrating growths which affect the entire organ. In two specimens only does the lesser curvature entirely escape. Of growths localised around the œsophageal opening we have three specimens, and all of these are spheroidal carcinomata. Of growths affecting the whole organ seven are spheroidal carcinomata, and one only is cylindrical carcinoma. We are thus led to conclude that there is nothing special in the character of the epithelium forming the mucous membrane or lining the glands at any part of the stomach, which renders it likely that a growth situated at that part should have the histological structure of one rather than of the other form of cancer. To this general statement there is a possible exception in the mucous membrane around the œsophageal orifice, in which position we have failed to find a limited growth of the cylindrical variety. As we have already pointed out, a diffuse infiltrating growth involving the whole organ is much more likely to be spheroidal than cylindrical carcinoma.

Turning now to the question of the sex and age of the patients, we have 36 cases available for this investigation. Taking all the cases of carcinoma together the proportion is 21 males to 15 females. In 43 cases noted by Dr. Fagge 30 were males, and 13 females. We have examined the post-mortem records for the 6 years, 1885 to 1890 inclusive, and find that of the 30 cases recorded in those years 20 were males and 10 were females. Of cylindrical carcinoma, 6 were males and 4 were females; of spheroidal carcinoma, 15 were males and 11 females. Grouping males and females together we find that the average age of all cases at the time of death was 52·1 years, the average in cases of cylindrical carcinoma being 52·6, and in spheroidal carcinoma 51·6 years. So far, therefore, as so small a number of cases avail for settling the question, it would seem that neither sex nor age has any determining influence on the character of the growth.

To ascertain the frequency and situation of secondary deposits thirty-four cases of carcinoma are available, which with four cases of sarcoma are tabulated below.

TABLE *of Malignant Growths of the Stomach, 34 Carcinomata, 4 Sarcomata, with secondary deposits.*

Nature of growth.	Sex.	Age.	Lymphatic glands.	Peritoneum.	Liver.	Lungs.	Pleura.	Spleen.	Supra-renal.	Kidney.	Intestine.	Ovary.	Bones.	No. of Case.	Remarks.
Cylindrical carcinoma.	M.	72	...	—	...	...	—	...	—	...	...	...	...	1	
	M.	58	—	...	...	—	...	...	...	...	...	...	...	11	Colloid.
	M.	50	—	...	...	...	...	...	...	...	...	...	...	10	
	M.	38	—	—	—	—	—	...	—	—	...	...	—	6	
	M.	65	...	...	—	...	...	...	...	...	...	...	...	12	
	M.	62	...	...	...	...	...	...	...	...	...	...	...	8	
	F.	56	...	...	—	...	...	...	...	...	...	...	...	7	
	F.	45	...	...	—	...	—	—	...	...	...	...	...	4	
	F.	47	...	...	...	...	...	...	...	...	...	...	...	5	
	F.	33	—	...	—	...	...	...	...	...	...	—	...	9	
Spheroidal carcinoma.	M.	48	—	...	...	...	...	...	...	...	...	...	...	40	
	M.	47	—	—	...	...	...	...	...	...	—	...	...	24	Colloid.
	M.	57	...	...	—	...	...	...	...	...	...	...	...	30	Colloid.
	M.	71	—	—	...	...	...	...	...	...	...	...	...	32	
	M.	37	...	—	...	...	...	...	...	...	...	...	...	33	
	M.	77	—	...	...	...	...	...	...	...	...	...	...	34	
	M.	67	—	...	...	...	...	...	...	...	...	...	...	23	
	M.	49	...	...	—	...	...	...	...	...	...	...	...	25	
	M.	45	...	...	—	...	...	...	...	...	...	...	...	27	
	M.	67	—	...	...	...	...	...	...	...	—	...	...	18	
	M.	43	...	...	—	—	...	—	...	...	...	...	...	38	
	M.	62	...	...	...	...	...	...	...	...	...	...	...	16	
	M.	47	—	—	...	—	—	...	...	...	...	...	...	37	
	F.	39	...	...	...	...	...	...	...	...	...	...	...	22	
	F.	65	—	...	...	...	...	...	...	...	...	...	...	13	
	F.	81	...	...	...	...	...	...	...	...	...	...	...	26	Colloid.
	F.	32	...	...	—	...	...	...	...	...	...	...	...	43	Colloid.
	F.	37	...	...	...	...	...	...	...	...	—	...	...	31	Colloid.

Nature of growth.	Sex.	Age.	Lymphatic glands.	Peritoneum.	Liver.	Lungs.	Pleura.	Spleen.	Supra-renal.	Kidney.	Intestine.	Ovary.	Bones.	No. of Case.	Remarks.
Spher. carc.	F.	34	...	...	...	...	...	...	...	...	...	...	...	14	
	F.	66	...	...	—	—	...	...	...	...	...	...	—	21	
	F.	45	...	—	—	...	...	...	...	...	...	...	...	44	Colloid.
	F.	33	...	—	...	...	...	...	...	...	...	—	...	28	
	F.	44	—	...	...	...	...	...	...	...	...	...	...	35	
	F.	47	—	...	...	...	...	...	...	...	...	...	...	19	
Sarcoma.	M.	67	...	—	...	...	...	...	—	...	...	...	...	46	
	M.	15	—	...	...	...	...	...	...	—	...	...	...	48	
	F.	38	...	...	...	...	...	...	...	...	...	...	...	45	
	F.	18	—	...	...	...	...	...	...	...	...	—	...	47	

From the above table it appears that secondary deposits are absent in 3 out of 10 cases of cylindrical carcinoma, and in 4 out of 24 cases of spheroidal carcinoma, in all, in 7 cases out of 34, or about 20 per cent. Other writers have given the percentage as 30 (*e. g.* Dr. Moore in his work on Medical Pathology, p. 289). There is no important distinction to be made out between the two classes of cancer, as regards the organs affected with secondary deposit. Lymphatic glands, liver, and peritoneum are affected most frequently, and probably in the order named. Our own table gives the proportion as 14, 12, and 8, while Dr. Moore in 29 cases finds it to be 18, 13, and 6. With regard to colloid degeneration it appears to be without influence on the situation or frequency of secondary deposits, for whilst in 10 cases of cylindrical carcinoma the total number of organs containing secondary deposits was 22, in the single colloid case two organs were thus affected. Similarly, in the spheroidal class 6 colloid cases presented secondary deposits in 8 organs, whilst in the whole series of 24 cases the organs affected by secondary deposit were 33 in number. The proportion is therefore practically the same. Contrary to the statements of some authors, our table seems to show that in colloid cases the liver is not affected less often and the peritoneum not more often than in cancers which have not undergone this change.

Before leaving the subject of carcinoma of the stomach, we should like to refer to some points which we have been able to investigate in relation to stenosis of the pylorus, growths limited to the cardia, and general induration and contraction of the organ.

Stenosis of the pylorus.—We have excluded from our consideration cases in which the whole organ is contracted, and cases in which the stenosis is produced by a growth which extends at the same time beyond the pyloric area. Of specimens in which the main feature is a narrowing of the pyloric orifice with more or less dilatation or hypertrophy of the stomach we have recently examined thirteen examples. They may be conveniently grouped under three headings: stenosis as the result (1) of irritant poisoning, (2) of cicatricial contraction consecutive to a chronic ulcer, and (3) of infiltration by new growth. We have failed to find any instance of pyloric obstruction, which could not be referred to one or other of these conditions.

Of stenosis due to corrosion we have two examples (Cases 53, and 54), and it is noteworthy that in one of these the history of irritant poisoning was not obtained till after the patient's decease, a circumstance which would seem very likely to occur when the poison has been taken with suicidal intent. The fact that the pyloric end only should be contracted in these specimens is in accord with the partial manner in which the mucous membrane is found to be destroyed in the rapidly fatal cases of corrosive poisoning.

Four examples of chronic ulcer of the pylorus (Cases 55, 56, 57, 13), leading to stenosis have been investigated. In one of these (Case 55) a similar ulcer at the middle of the lesser curvature has led to a marked hour-glass contraction of the organ, and in another (Case 13) the indurated edge of the ulcer is infiltrated with a growth of spheroidal carcinoma. This last specimen raises the interesting question of the relation of carcinoma to chronic ulceration, which, though illustrated by several other of our preparations, is too large a subject to be entered upon in this paper.

In the remaining cases, twelve in number, there is a localised growth surrounding the pyloric end of the stomach, and causing obstruction either by contracting the orifice or by projecting prominently above the mucous surface. In five instances the growth is a spheroidal carcinoma, in five it is a cylindrical carcinoma, and in two it is a round-celled sarcoma. In one of the cases (No. 19) of spheroidal carcinoma the thickening of the wall was found after repeated examination under the microscope to be due to a deposit of well-formed fibrous tissue in which no epithelioid cells were apparent. In the former catalogue this specimen was described as a spindle-celled sarcoma. Histological examination of a slightly enlarged neighbouring lymphatic gland showed it to contain epithelioid cells arranged in alveolar spaces, and hence we have concluded the original growth to be a scirrhous spheroidal carcinoma. We were strengthened in this conclusion by having observed that in some of the specimens of general neoplastic induration epithelial elements could not be found in the thick fibrous tissue replacing the submucosa, though they were readily recognised in the lymphatic glands of the greater or lesser curvature. We would suggest that such cases as these may account for at any rate a considerable number of the examples of so-called non-malignant scirrhous pylorus, due neither to corrosive poisoning, nor to the contraction following a chronic ulcer.

Growths limited to the cardia.—A few words are necessary with reference to the circumscribed growths at the œsophageal orifice, which, as we have seen in our three specimens, have the histological characters of spheroidal carcinoma. Dr. Fagge writes (' Principles and Practice of Medicine " ed. 1, vol. ii, p. 146) : "It is generally said that there are three distinct varieties of this disease (*sc.* cancer of the stomach) respectively affecting the cardiac orifice, the body of the stomach, and the pyloric orifice. Now, as regards the first of these, I may at once state my opinion that almost all the cases which have been set down as examples of cancer affecting the cardia have really been instances of cancer of the end of the œsophagus extending into the adjacent part of the stomach." "So far as I can discover," says Dr. Fagge, "the 'Pathological Transactions' contain not a single example of cancer beginning in the stomach at its œsophageal end, nor does the Museum of Guy's Hospital show any such specimen, in which the lower end of the œsophagus is not also affected." As will be seen by reference to our plate this last statement is quite correct. Nevertheless we venture to think that Dr. Fagge was mistaken in his view that these growths really arise from the œsophagus, a view which it is probable that he adopted because "on *a priori* grounds we should expect that a part at which the digestive tube is opening out into a large cavity should have little or no tendency to be affected with the disease in comparison with the narrow passage above it." We know so little of the cause of cancer anywhere that, for our part, we cannot give much weight to this *a priori* consideration, especially as a similar line of argument would lead to the conclusion that cancer at the lower end of the ileum was much more common than in the cæcum, a conclusion which would be opposed to all experience. Our own belief that these growths really arise from the stomach rests upon the following experience.

We have examined microscopically more than thirty specimens of malignant growths from all parts of the œsophagus, and with the exception of two cases of sarcoma, every growth so examined proved to be a squamous epithelioma. The only growth on our shelves which we decided upon microscopic evidence to be a spheroidal carcinoma we found subsequently to be situated at the extreme limit of the œsophagus, and involving the cardia. With reference, therefore, to the three specimens of spheroidal carcinoma mentioned above it appears to us more reasonable to believe that they are gastric growths than that they are œsophageal in origin, as Dr. Fagge thought. If his view be correct, it remains to be explained why they alone among such growths are spheroidal, whilst at every other part of the tube nothing but squamous epithelioma is to be found. It is perhaps well to emphasise our agreement with Ziegler that "primary cancer of the œsophagus is a squamous epithelial carcinoma," when we find in a recent work on medical pathology the statement that "new growth in the œsophagus is generally carcinoma with much stroma and flat spheroidal or columnar cells in the meshes of the stroma."

Induration of the stomach (*india-rubber bottle stomach*).—In ten of the specimens examined the wall of the stomach was found to be universally indurated, and in most of these specimens the cavity of the viscus was contracted. In two only of these cases (Nos. 51 and 52) were we able to ascribe the condition to any other cause than carcinomatous infiltration. In one of these non-malignant cases (No. 51) there was a definite history of the patient having swallowed corrosive poison three months before her death, and in this preparation there was a perforation in the wall of the organ communicating with a small abscess cavity between the spleen and the diaphragm. No history of poisoning was recorded in the other case, though the replacement of a large portion of the mucous membrane by dense cicatricial tissue suggests such an origin for the condition. In both stomachs the contraction was extreme, the former measuring only five inches along its greater curvature, and the latter barely holding an ounce of fluid.

Of the eight specimens (Cases 10, 29 to 35) in which the condition is due to malignant growth, the smallest measures seven inches along its greater curvature, and two inches transversely at its widest part, whilst some of them show little if any diminution in size. In one example only (Case 10), and this is a specimen in which the organ is of its normal size, an infiltrating growth has the characters of a cylindrical carcinoma. The stroma is scanty, and, as is usual in medullary growths, the muscular coat is considerably invaded.

The growth which has produced the induration in the remaining seven specimens is a spheroidal carcinoma, the amount of fibrous stroma varying generally with the degree of contraction of the organ. Histologically, the most marked characteristic of the growth in these cases is the extreme irregularity in the size and shape of the alveoli, and the scantiness of epithelioid elements, so that it was often necessary to examine several sections before we could satisfy ourselves of the carcinomatous nature of the deposit. Indeed, in two cases the true character of the disease was revealed only by the existence of a secondary deposit of spheroidal carcinoma in the neighbouring lymphatic glands. On this point we agree with Ziegler that "cases occur in which no cell-nests are to be made out even when the tissue is microscopically examined, and then the only evidence forthcoming as to the cancerous nature of the disease may be the metastatic growths to which it has given rise."

Taken as a class, these cases of malignant induration of the whole organ show but little ulceration of the mucous membrane, which is usually smooth even in the smallest stomachs. The submucosa is in all cases thickened, and is commonly thicker towards the pyloric end than at the fundus. It is, however, noticeable that the wall of the smallest stomach is only about one eighth of an inch in thickness, whilst in some of larger size it is as much as half an inch. Part of this increase of thickness is due to hypertrophy of the muscular coat, which to the naked eye shows little sign of infiltration, but under the microscope is found to be traversed by numerous septa, in some of which epithelioid cells can be discerned. The serous coat is also somewhat thickened and opaque. In the two cases (Nos. 30 and 31) in which colloid degeneration is observed under the microscope, the macroscopical characters of the growth are such as we have previously described as produced by this change.

Seven of the patients from whom these specimens were taken were males and two were females ; their average age at death did not materially differ from the average in all cases of carcinoma. On reference to the table it may be seen that in Case 10 there were secondary deposits in the liver, and in each of six others available for this investigation secondary deposits were found in one organ.

Under the heading of chronic induration of the stomach some authors have described what they believe to be a condition dependent on chronic inflammation or cirrhosis, and not arising from malignant disease. In the former catalogue of the Museum two of the specimens we now recognise as carcinomatous were thus described, and one of them is fully reported in the thirteenth volume of the 'Transactions of the Pathological Society' (p. 83). For our own part we are persuaded that this condition of induration, except when due to traumatism, should be invariably ascribed to malignant growth, though, as we have before said, it may require very careful examination to establish the fact.

Sarcoma of the stomach.—Of this very rare disease we have four examples, all of them round-celled sarcomata with a remarkable uniformity of minute structure (Cases 45 to 48). The mucous membrane in the sections examined was but slightly affected with growth, while the submucosa was greatly thickened and infiltrated with round cells, which extended into the muscular coat, causing a very complete destruction of its proper tissue. The serous coat was thicker than normal, and showed an increase of fibrous tissue, in which were seen numerous sarcomatous cells. In contrasting these specimens with examples of lymphadenoma of the stomach, we notice in them that the cells are larger, less uniform in size and shape, and that there is a much greater amount of fibrous tissue in the submucosa. Moreover, in lymphadenomata the muscular and serous coats are free from infiltration. We have no specimen of spindle-celled sarcoma, and, as we have already pointed out, the preparation described as such in the former catalogue we have included amongst the scirrhous spheroidal carcinomata on the evidence of the secondary deposit found in an enlarged gland.

In accordance with the histological appearance above described we find that the mucous membrane in these specimens is smoothly stretched over the growth, and that it is little if at all ulcerated; that the growth is for the most part in the submucosa, which in some cases is half an inch in thickness, and that the muscular coat is sometimes so much invaded that it is impossible to recognise it as a separate layer in a section through the wall of the organ. In this last respect the sarcomata differ from diffuse infiltrating carcinomata, in which the hypertrophied muscular coat often contrasts strongly with the white growth in the submucosa. In three of the cases the growth produces a smooth thick plaque, the edge of which is abrupt and clearly defined from the adjacent healthy wall. In the remaining specimen a smooth globular mass about the size of a pigeon's egg projects into the stomach from the surface of such a plaque. As shown in the diagrams in Plate I we notice that, as with other malignant growths, the pylorus is most commonly affected, and that the fundus of the organ escapes. The most distinguishing fact with regard to localisation is that in two out of the four cases of sarcoma (Cases 47 and 48) the growth extends into the duodenum. In all our specimens of carcinoma involving this part of the organ the growth is sharply limited by the pyloric ring. In our experience an india-rubber bottle stomach is never sarcomatous.

Two of the patients from whom these specimens were taken were males and two females, and their ages were 15, 67, 18, and 38, respectively. As shown in the table, secondary deposits of growth were present in three of the cases.

Appendix.

We have appended a short history of the cases and description of the specimens referred to in the above paper.

Case 1.—John D—, æt. 72, under Dr. Bright. Admitted for phthisis and dyspepsia. There was a large irregular ulcer on the posterior wall of the stomach near the pylorus. The edge of the ulcer was thickened, and infiltrated with cylindrical carcinoma. There were secondary deposits in the pleura, peritoneum, and one of the suprarenals. New Catalogue, No. 3698.

Case 2.—A specimen of a stomach showing a raised and deeply ulcerated mass of new growth encircling the pylorus, and extending for two and a half inches into the body of the organ. The growth, which is a cylindrical carcinoma, is beset with numerous villous processes, and projects from the serous surface. New Catalogue, No. 3702.

Case 3.—A specimen of a somewhat dilated stomach, the pyloric end of which is narrowed by a thick deposit of cylindrical carcinoma, which extends for three and a half inches into the body of the organ. New Catalogue, No. 3703.

Case 4.— Susan V—, æt. 45, under Dr. Bright. Admitted for vomiting. She was ill for five months. There is an ulcer two inches in diameter close within the pylorus. The edge is raised, and infiltrated with a growth of cylindrical carcinoma. The pyloric orifice is narrowed and is surrounded by several enlarged caseous lymphatic glands.

There were secondary deposits in the pleuræ, liver, and spleen. New Catalogue, No. 3704.

Case 5.—Sarah C—, æt. 47, under Dr. Fagge. Admitted for gastric pain and vomiting, with a well-defined tumour in the abdomen. She was ill for fourteen months. There was a deep ulcer on the posterior wall of the stomach just within the pylorus. Tho wall of the organ around was thickened, and infiltrated by a growth of cylindrical carcinoma. The pyloric orifice was narrowed. New Catalogue, No. 3705.

Case 6.—Job S—, æt. 38, under Dr. Goodhart. Admitted for vomiting and tumour in the umbilical region. He was ill for twelve months. The whole stomach, except a small portion at the cardiac end, was infiltrated with cylindrical carcinoma. There was a large mass of growth projecting into the organ about its middle third. There were secondary deposits in the skull, sternum, and most of the viscera. New Catalogue, No. 3701.

Case 7.—Elizabeth C—, æt. 56, under Dr. Rees. Admitted for gastric pain and occasional hæmatemesis. There was a mass of villous growth with raised everted edges upon the lesser curvature, and part of the anterior and posterior wall of the stomach. The liver was directly invaded. Histologically the growth is a cylindrical carcinoma. New Catalogue, No. 3697.

Case 8.—Job S—, æt. 62, under Dr. Habershon. Admitted for gastric symptoms. On the lesser curvature and on the anterior and posterior wall of the stomach there was an ulcerated growth reaching from the œsophageal opening to within two and a half inches of the pylorus. The ulceration has exposed a portion of the pancreas to which, as well as to the liver, the stomach was bound by adhesions. Histologically the growth is a carcinoma, transitional between cylindrical and spheroidal carcinoma. New Catalogue, No. 3711.

Case 9.—Helen W—, æt. 33, under Dr. Rees. Admitted for vomiting. She was ill for eighteen months. There was a deep ulcer upon the lesser curvature the margin of which was infiltrated by cylindrical carcinoma. There were secondary deposits in the liver, one ovary, and lymphatic glands. See Insp., 1858, No. 140.

Case 10.—Mr. W—, æt. 50, suffered for some months with constant vomiting and emaciation. The walls of the stomach were throughout infiltrated with a diffuse growth of cylindrical carcinoma. There was an irregular ulcer upon the lesser curvature. Secondary deposits in the lymphatic glands. New Catalogue, No. 3700.

Case 11.—Joseph L—, æt. 58, under Dr. Wilks. Admitted for gastric pain, cough, and hæmoptysis of four months' duration. There was a rounded mass of growth three inches in diameter projecting from the posterior wall of the stomach. The surface was ulcerated, had a gelatinous appearance, and in structure was a cylindrical carcinoma undergoing colloid change. New Catalogue, No. 3699.

Case 12.—Martin F—, æt. 60, under Dr. Gull. Admitted for vomiting and wasting. He was ill for sixteen months. Near the pylorus there was a small ulcer, the raised edges of which were infiltrated with cylindrical carcinoma. There were secondary deposits in the liver. See Insp., 1856, No. 226.

Case 13.—Elizabeth R—, æt. 65, under Dr. Pavy. Admitted for pain after food and vomiting, from which she had suffered more or less for the greater part of her life. At the autopsy the pyloric orifice was found to be obstructed, and there was a small oval ulcer on the lesser curvature just within the pyloric ring. The edges of the ulcer were sharply defined, and the surrounding tissues were infiltrated with spheroidal carcinoma. New Catalogue, No. 3677.

Case 14.—Emma P—, æt. 34, under Dr. Lever. Admitted for puerperal pyæmia. Just within the pylorus a small area of the mucous membrane presented a raised polypoid appearance without any ulceration. The submucous tissue was infiltrated with spheroidal carcinoma, which did not invade the muscular coat. There were no secondary deposits. New Catalogue, No. 3719.

Case 15.—William W—, who died from ascites. Upon the posterior wall of the stomach just within the pylorus there was a large bossy tumour projecting into the stomach, over which the mucous membrane was smoothly stretched. In the central part of the mass there was an oval ulcer. Histologically the growth is a spheroidal carcinoma. New Catalogue, No. 3721.

Case 16—.John L—, æt. 62, under Dr. Barlow. There was a raised warty growth of spheroidal carcinoma limited to the pyloric end of the stomach. There were no secondary deposits. See Inspection Book, vol. xxxii, p. 215.

Case 17.—A specimen of a greatly hypertrophied and dilated stomach, the pyloric extremity of which is encircled and narrowed by a growth of colloid spheroidal carcinoma, which extends inwards from the duodenum for two and a half inches. New Catalogue, No. 3723.

Case 18.—John T—, æt. 67, under Dr. Hughes. Admitted for gastric pain, and a tumour in the region of the stomach. He had had dyspepsia for one year. There was a circular mass of growth extending from the pyloric ring along the greater curvature of the stomach. The surface of the growth was sloughy, and there was a communication between it and the transverse colon. The growth was a spheroidal carcinoma, and had produced secondary deposits in the mesenteric glands. There was a similar growth in the cæcum. New Catalogue, No. 3727.

Case 19.—Rachel B—, æt. 47, under Dr. Habershon. Admitted for vomiting, from which she had suffered for twelve months. The stomach was greatly hypertrophied and dilated, and its pyloric extremity was encircled by a growth which extended from the duodenum four inches into the body of the organ. The mucus membrane was stretched over the mass, the margin of which was well defined. The growth was situated in the submucous tissue, and was in parts half an inch thick. Histological examination of the adventitious material showed it to consist of well-formed fibrous tissue, while in the periphery of an adjacent lymphatic gland there were found small alveoli filled with spheroidal epithelioid cells. There were no other secondary deposits. New Catalogue, No. 3723A.

Case 20.—A specimen of a stomach showing the pyloric extremity encircled and narrowed by a growth of colloid spheroidal carcinoma, which extends from the duodenum five inches towards the fundus of the organ. There were secondary deposits in the glands of the less curvature. New Catalogue, No. 3722.

Case 21.—Mary H—, æt. 66, under Mr. Hilton. Admitted for spontaneous fracture of the femur. There was a nodulated mass of spheroidal carcinoma in the mucous membrane of the pyloric half of the stomach, with secondary deposits in the liver, lungs, and femur. See Inspection Book, 1861, No. 97.

Case 22.—Isabel H—, æt. 39, under Dr. Gull. Admitted for gastric pain and vomiting. She was ill for ten months. The stomach was found to be firmly adherent at its pyloric end to the abdominal wall. There was a cicatrised ulcer completely encircling the organ one inch within the pylorus. The edge of the ulcer was infiltrated with spheroidal carcinoma. There were no secondary deposits. New Catalogue, No. 3676.

Case 23.—Joseph P—, æt. 67, under Dr. Taylor. Admitted for pain after food, vomiting, and with a tumour on the right side of the abdomen. He was ill five months. The stomach was found to be dilated, and there was a large flat mass of spheroidal carcinoma occupying the entire circumference of the organ, and extending from the pylorus about four inches towards the cardia. The mass had a well-defined edge, and its surface was in parts ulcerated. There were secondary deposits in the lymphatic glands. New Catalogue, No. 3720.

Case 24.—Jonn C—, æt. 47, under Dr. Barlow. Admitted for vomiting and an abdominal tumour. He was ill for ten weeks. There was an infiltrating growth involving the lesser curvature of the stomach along its whole length, and extending along both surfaces of the organ almost as far as the greater curvature. Histologically the growth is a spheroidal carcinoma undergoing colloid change. There were secondary deposits in the lymphatic glands and the peritoneum; and there was a malignant stricture of the rectum. New Catalogue, No. 3710.

Case 25.—James S—, æt. 49, under Dr. Wilks. Admitted for hæmatemesis and gastric pain. There was a growth of spheroidal carcinoma encircling the pyloric extremity of the stomach. The gastric veins along the lesser curvature were distended by cancerous thrombus, which had spread into the portal vein, and thus produced secondary deposits in the liver. New Catalogue, No. 3724.

Case 26.—Elizabeth P—, æt. 81, under Mr. Davies-Colley. Admitted for a strangulated femoral hernia, from which she died. There was no previous history of any disease of the stomach. There was a slightly raised ulcerated growth extending from within the pyloric ring along the lesser curvature for a distance of five inches, and affecting both walls of the organ so as nearly to reach the greater curvature. The surface presented numerous gelatinous-looking nodules, and histologically the growth was a colloid spheroidal carcinoma. New Catalogue, No. 3712.

Case 27.—Philip S—, æt. 45, under Dr. Wilks. Admitted for anorexia and vomiting. He died suddenly from gastric hæmorrhage. An ulcerated growth was found in the stomach upon the lesser curvature. In the sloughing base of the ulcer, the splenic artery was discovered opened by ulceration. The growth was a spheroidal carcinoma. New Catalogue, No. 3725.

Case 28.—Mary C—, æt. 33, under Dr. Rees. Admitted for gastric rain and vomiting with hæmatemesis. She was ill for two years. There was a diffuse infiltrating growth of spheroidal carcinoma occupying the middle third of the stomach. There was secondary deposits in the peritoneum and in the ovaries. Inspection Book, 1886, No. 137.

Case 29.—A man, æt. circa 53. He was a drunkard, and suffered from vomiting and dysphagia. The cavity of the stomach was extremely contracted, and its coats much thickened. The submucous tissue was infiltrated with a growth of spheroidal carcinoma. Old Catalogue, No. 1794.

Case 30.—William C—, æt. 57, under Dr. Bright. Admitted for nausea and pain in the epigastrium. He was ill for five months. The stomach was somewhat contracted, and thickened throughout by a gelatinous deposit of spheroidal carcinoma with much colloid change. Secondary deposits in the liver. New Catalogue, No 3714.

Case 31.—Elizabeth T—, æt. 37. Admitted for pain after food with emaciation. She was ill for five months. The stomach was somewhat contracted, and its walls universally thickened by a deposit of colloid spheroidal carcinoma, chiefly in the submucosa. New Catalogne, No. 3715.

Case 32.—Joshua B—, æt. 71, under Dr. Habershon. Admitted for gastralgia. He was ill for six months, and at no time did he suffer from vomiting. The stomach measured 8½ inches along its greater curvature, and 2½ inches transversely. The walls were thickened throughout, partly by a deposit of spheroidal carcinoma chiefly in the submucosa, and partly by muscular hypertrophy. There were secondary deposits in the gastric glands and in the peritoneum. New Catalogue, No. 3716.

Case 33.—Joseph R—, æt. 37, under Dr Pye-Smith. Admitted for vomiting and ascites, and a recent attack of hæmatemesis. The stomach measured eight inches along its greatest curvature, and two and a half inches transversely at its widest part. The wall was universally thickened, and the mucous membrane slightly ulcerated. All the coats of the organ were infiltrated by spheroidal carcinoma. There were secondary deposits in the omentum. New Catalogue, No. 3717.

Case 34.—A male, æt. 77, who suffered from gastric pain, vomiting, and emaciation, A tumour was felt nine months before death. The stomach measured seven inches along its greater curvature, and two inches transversely at its widest part. The walls were three eighths of an inch thick, and there was a deposit in the submucosa of spheroidal carcinoma, which extended also into the other coats. There were secondary deposits in the lymphatic glands. New Catalogue No. 3718.

Case 35.—Mary F—, æt. 44, under Dr. Rees. Admitted for ascites with no symptoms of disease of the stomach. The stomach was somewhat contracted and its walls thickened throughout, especially towards the pylorus. The mucous membrane was not ulcerated. Under the microscope there was an excess of fibroid material in the submucosa with no clear evidence of growth, but in an enlarged adjacent lymphatic gland there was a deposit of spheroidal carcinoma. New Catalogue, No. 3718a, and 'Trans. Path. Soc.,' vol. 13, p. 83.

Case 36.—A specimen of a stomach showing a rounded ulcer occupying the œsophageal half of the lesser curvature. The ulcer has a sloping margin, and its base presents a reticulated appearance. The thickened wall of the stomach is infiltrated with a growth of spheroidal carcinoma undergoing colloid degeneration. New Catalogue, No. 3709.

Case 37.—Henry N—, æt. 47, under Dr. Goodhart. Admitted for epigastric pain, and a tumour in the left hypochondriac region, with occasional hæmatemesis. He was ill for five months. There was a limited growth of medullary spheroidal carcinoma surrounding the œsophageal opening of the stomach and extending along the lesser curvature. There were secondary deposits in the peritoneum, pleuræ, lungs and mediastinal glands. Inspection Book, 1890, No. 189.

Case 38.—Daniel C—, æt. 43, under Dr. Gull There was a diffuse infiltration of the lesser curvature of the stomach extending into the œsophagus. The surface was ulcerated and histologically the growth was a spheroidal carcinoma. There were secondary deposits in the liver, spleen, and lungs, Inspection Book, 1861, No. 244.

Case 39.—A specimen of a stomach showing the mucous membrane covering the cardia and fundus of the organ to be hypertrophied and markedly rugose, while the submucous tissue is infiltrated with a growth of spheroidal carcinoma. New Catalogue, No. 3708.

Case 40.—Richard W—, æt. 48, under Mr. Bryant. Admitted for dysphagia of four months' duration. The stomach was attached to the abdominal wall as a preliminary step to the operation of gastrostomy, but as the patient was afterwards able to swallow food more easily, the organ was not opened. There was a small growth encircling the œsophageal orifice of the stomach, and invading the last inch of the gullet. Histologically it is a spheroidal carcinoma. New Catalogue, No. 3706.

Case 41.—A specimen of a stomach showing the œsophageal orifice surrounded and somewhat obstructed by an irregular mass of growth. The edges of the growth are thick, and the central portion is deeply ulcerated. Histologically it is a spheroidal carcinoma. New Catalogue, No. 3707.

Case 42.—A specimen of spheroidal carcinoma of the cardiac end of the œsophagus, which extends for a short distance into the stomach. There is deep ulceration, which communicates with the right lung. No clinical account preserved. New Catalogue, No. 3597.

Case 43.—Eleanor M—, æt. 32, under Dr. Barlow. Admitted for vomiting and anæmia. There had been a prominent abdominal tumour "for a long time." The wall of the stomach was in large part destroyed and replaced by a mass of colloid growth, which freely invaded the abdominal wall and the neighbouring viscera. There was a large secondary deposit in the liver. Histologically the growth was a spheroidal carcinoma with scanty stroma and colloid change. New Catalogue, No. 3713.

Case 44.—Maria C—, æt. 45, under Mr. Bryant. Admitted for syphilitic stricture of the rectum. She had suffered for three months from gastric pain and jaundice. There was a large ulcer on the lesser curvature of the stomach, and the wall of the organ throughout was thickened by a deposit of colloid spheroidal carcinoma. There were secondary deposits in the mesentery, omentum, and liver. See Inspection Book, 1888, No. 135.

Case 45.—Louisa B—, æt. 38, under Dr. Goodhart. Admitted for vomiting, diarrhœa, and distension of the abdomen. She was ill for six weeks. There was a globular mass of round-celled sarcoma about the size of a pigeon's egg, projecting upon the posterior wall of the stomach near the pylorus. Around the mass the walls of the organ were thickened by extension of the growth, the thickening being separated by a well-defined margin from the healthy parts beyond. There were numerous peritoneal adhesions but no secondary deposits. New Catalogue, No. 3731.

Case 46.—Thomas S—, æt. 67, under Dr. Barlow. Admitted for symptoms of malignant disease of the stomach. The lesser curvature of the stomach throughout its length was occupied by a mass of growth, which appeared as a thick plaque towards the pyloric end. Histologically it was a round-celled sarcoma, and had produced secondary deposits in the peritoneum, and the right supra-renal capsule. The original growth involved the pancreas and the neighbouring lymphatic glands. New Catalogue, No. 3730.

Case 47.—Elizabeth S—, æt. 18, under Dr. Addison. Admitted for frequent vomiting and a tumour in the abdomen. She was ill for five months. The stomach was considerably dilated, and showed a flat mass of growth occupying its pyloric half and extending for a short distance into the duodenum. Towards the œsophageal orifice the end of the growth was abrupt, whilst in the duodenum the limit is ill-defined. The submucous and muscular coats of the organ were replaced by a growth of round-celled sarcoma. There were secondary deposits in the gastric and lumbar glands and in both ovaries. New Catalogue, No. 3732.

Case 48.—Frederick W—, æt. 15, under Dr. Gull. Admitted for an abdominal tumour and jaundice. There was an infiltrating growth of round-celled sarcoma occupying the pyloric half of the stomach and extending about an inch into the duodenum. The mucous membrane covering the growth was mostly smooth, though in parts ulcerated. There were secondary deposits in the mesenteric glands and in one of the kidneys. New Catalogue, No. 3733.

Case 49.—Isabella D—, æt. 65, under Dr. Addison for cirrhosis of the liver. There were no gastric symptoms. At the autopsy large masses of soft shaggy growth were found projecting into the cavity of the stomach. They were attached along the greater curvature and anterior wall of the organ. Except at the point of attachment the coats of the organ are normal, and under the microscope there was no evidence of growth. New Catalogue, No. 3694.

Case 50.—A man, æt. 40, who suffered from anæmia for eleven months. No history of vomiting or hæmorrhage. There was a cauliflower-like mass of growth projecting from the posterior wall of the stomach. No evidence of malignancy. New Catalogue, No. 3695.

Case 51.—Sarah R—, æt. 22, under Dr. Wilks. Admitted for vomiting, having three months previously swallowed some Burnett's Disinfecting Fluid. She died from pneumonia and peritonitis, and at the autopsy the stomach was found to be much contracted, measuring only five inches along its greater curvature. The mucous membrane was ulcerated, and the wall perforated in two places. New Catalogue, No. 3641.

Case 52.—Thomas F—, æt. 34, under Dr. Habershon.

Admitted for regurgitation of food and emaciation. He was ill eight months. No history of irritant poisoning. The stomach was so contracted as barely to hold an ounce of fluid. The walls were half an inch thick, and there were cicatrices upon the mucous membrane. There were no secondary deposits, and the thickened wall showed no evidence of malignant growth. New Catalogue, No. 3651.

Case 53.—Charles C—, æt. 34, under Dr. Pavy. Admitted for hæmatemesis and dysphagia of ten weeks' duration. Duodenostomy was performed. After his death it appeared at the inquest probable that he had attempted to commit suicide by taking poison three months previously. At the autopsy the pylorus was extremely small, and the inner surface of the stomach along the lesser curvature was marked by scars. The lower end of the œsophagus was slightly contracted. New Catalogue, No. 3650.

Case 54.—Henry G—, æt. 43, under Dr. Hale White. Admitted for vomiting and dilatation of the stomach, following upon corrosive poisoning by spirits of salts seven weeks before admission. Gastrostomy was performed, and he was fed by means of a catheter passed into the duodenum. He died six days later, and at the autopsy the pylorus was found to be narrowed, and the walls of the stomach hypertrophied. There was no ulceration or scarring of the mucous membrane. New Catalogue, No. 3640, and the 'Transactions' of the Clinical Society for 1891.

Case 55.—Jane L—, æt. 32, under Dr. Hale White. Admitted for vomiting of several years' duration. There was obvious dilatation of the stomach. There were two non-malignant ulcers, one at the pylorus and the other about the middle of the lesser curvature. The contraction of the indurated wall in the neighbourhood of the ulcers had given rise to the narrowing of the pylorus and the hour-glass condition of the stomach. The pyloric pouch held ninety ounces and the œsophageal forty ounces of fluid. See Inspection Book, 1891, No. 301.

Case 56.—Mary W—, æt. 22, under Dr. Addison. Admitted for gastric pain and vomiting of three years' duration. A tumour was felt in the right hypochondriac region. At the autopsy the stomach was found to be much dilated, and there was a stricture of the pylorus two inches in length. In the middle of the constriction was a deep ulcer with smooth base and shelving edges. Histologically there is no evidence of malignant growth. New Catalogue, No. 3662.

CASE 57.—A specimen of the pyloric end of a stomach showing just within the pylorus two or three small deep ulcers. The wall of the organ in the neighbourhood of the ulcers is much thickened, and the pyloric orifice narrowed. Under the microscope there was no sign of malignant growth.

DESCRIPTION OF PLATES I, II, III, AND IV,

Illustrating Drs. Perry and Shaw's paper on Malignant Disease of the Stomach.

PLATE I.

Villous tumours, Cases 2 and 7, malignant; Cases 49 and 50, non-malignant. Sarcomata, Cases 45—48.

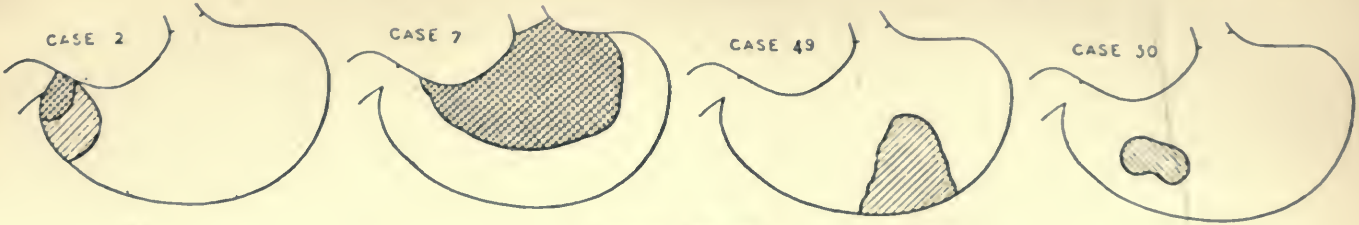
PLATE II.

Cylindrical carcinomata, Cases 1—11.

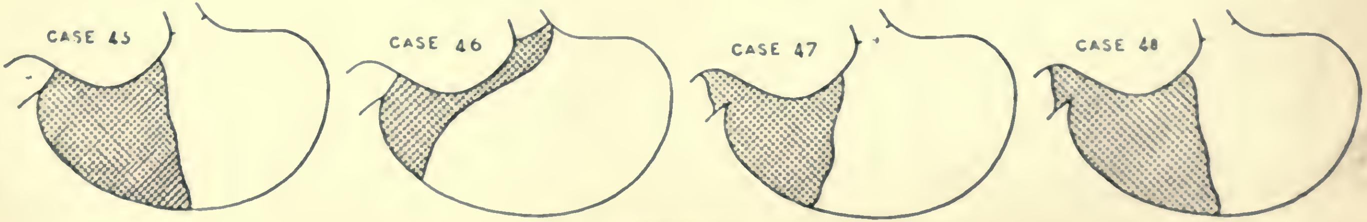
PLATES III AND IV.

Spheroidal carcinomata, Cases 13—42.

The extent of the growths on the anterior wall is shown by lines drawn downwards to the left, on the posterior wall by lines drawn downwards to the right.



VILLOUS TUMOURS.



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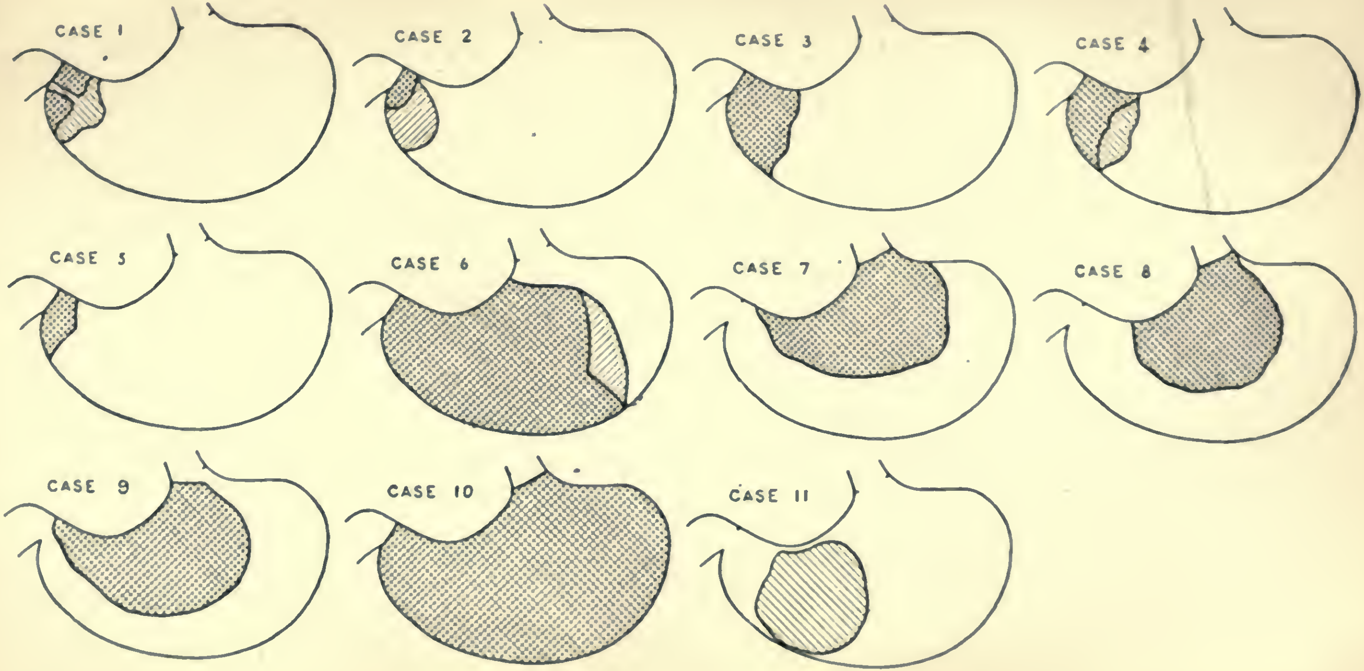

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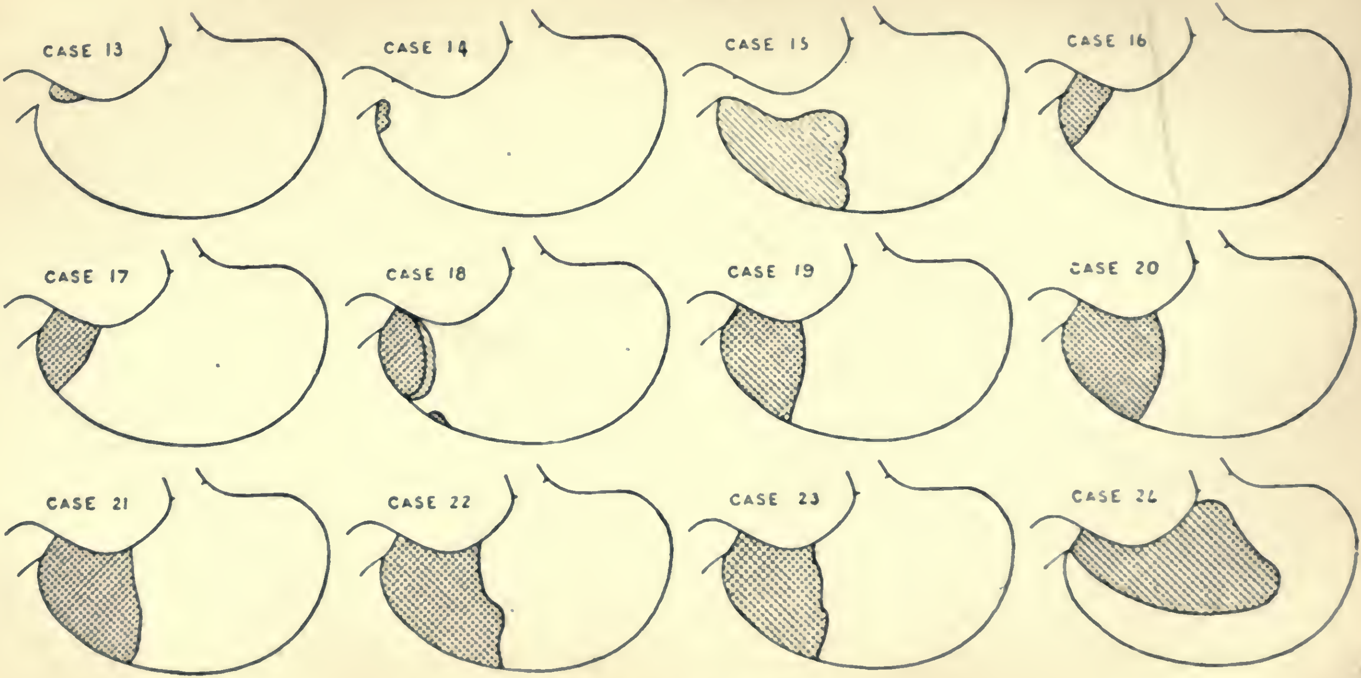

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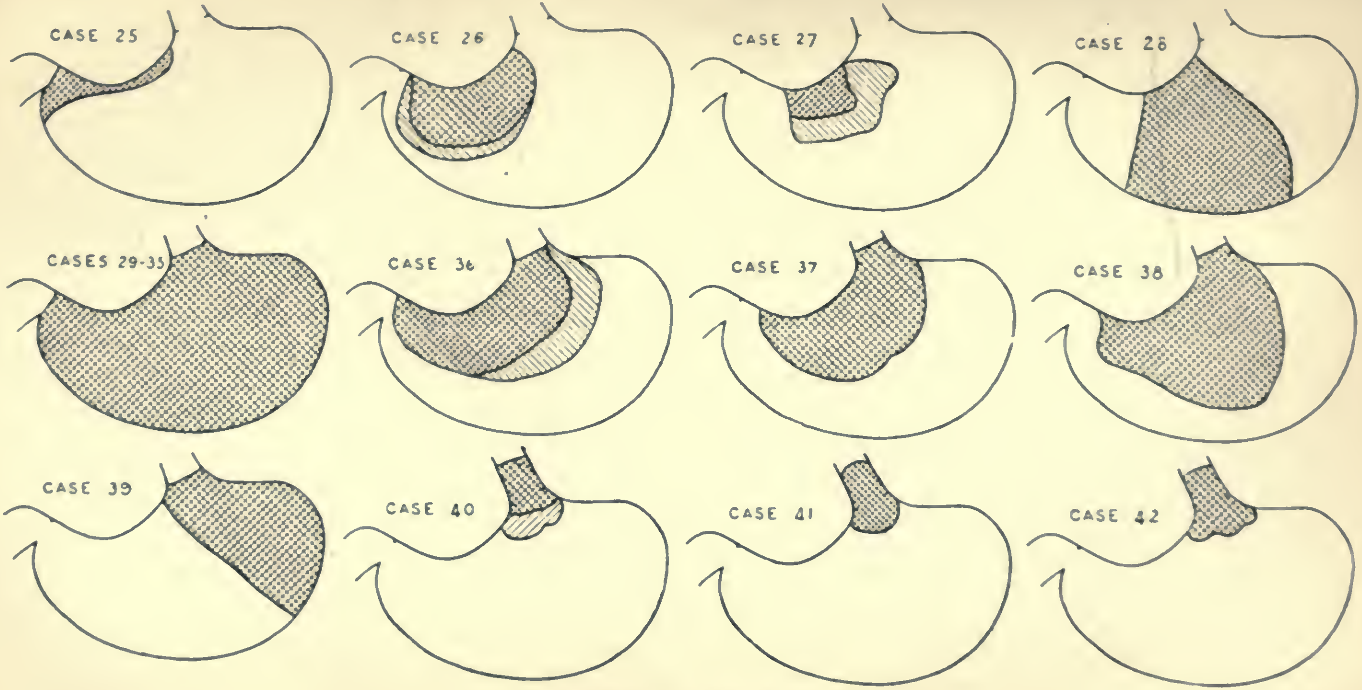

SPHEROIDAL CARCINOMATA.

A CASE OF PEMPHIGUS AFFECTING THE CONJUNCTIVA.

By W. A. BRAILEY.

The subject of this rare affection, T. A—, a gentleman of German extraction, æt. 64, came from the New England district of the United States in June, 1890. He was generally healthy and of spare active temperament. Though the conjunctival affection was the one for which he sought immediate relief, the soft palate, pharynx, larynx, nasal mucous membrane, and skin were then or had been recently involved.

He is inclined to attribute his pemphigus to the eating of raw apples in 1881, and he says that on two occasions after doing this he lost all power of moving his limbs.

In the autumn of 1883 his throat first became affected, and in January, 1884, his left nostril bled for some time, a thing which has continued ever since at occasional intervals. His eyes have been affected for six years, but did not trouble him much till September, 1889, when the inversion of the lids and consequent trichiasis became a constant cause of irritation. This ocular condition is clearly due to a shrinking of the conjunctival pouch, especially the lower, the mucous membrane of which when put on the stretch by gently pulling down the lower lid, has the appearance of a series of ridges with short intervening hollows, extending from the inner aspect of the lids to the globe. These ridges are most marked opposite the region of the inverted eyelashes, *i. e.* at the centre of each lower lid and towards the outer side of the left upper lid. When first seen, a bleb, the size of a large shot, was visible on the conjunctiva of the left upper lid, just outside its centre, not very far from its free margin.

The back of the pharynx is dry and yellowish brown in colour. There are about a dozen small blebs on the posterior part of the roof of the mouth. His nose troubles him, as it has done ever since it first bled, by a constant free discharge of mucus.

Exertion quickly brings out pemphigus vesicles on his soft palate, but his symptoms occur far more commonly in distinct relation to his diet. Raw fruit brings on pemphigus of the skin and other parts at once. Acid fruit generally is a poison, he says. A single strawberry or cherry is sufficient to bring out blebs within a few hours. Nuts or almonds will serve him in the same way, as will cheese. Most articles of diet have been forbidden him by different authorities or by his own experience. He lives mainly on soup, soft-boiled eggs, milk, rice, poultry, and white fish. Claret and light Rhine wines agree.

In 1886, Royat water, internally and in compresses, gave relief from the skin symptoms in a few hours; but its application by gargle or spray benefited neither throat nor nose.

Since then arsenic, in the form of mixture or pills, has generally benefited his skin, but lately it has caused him griping pains, and he will take it no more.

Extremely few cases have been recorded where pemphigus blebs have actually been seen on the conjunctiva. But some dozen have been published in which, together with pemphigus of the skin or mucous membranes, past or present, the conjunctival sacs have been shrunken. It seems reasonable to suppose that such are genuine cases of conjunctival pemphigus, and that previously existing blebs have been the cause of the shrinking.

But at least as many cases have been recorded in which the shrinking of the sac is the only symptom observed, and it is a question whether such can be associated with pemphigus.

Stellwag and von Graefe, who first described this condition, thought that it was an independent affection, and the latter applied to it the term "Essential shrinking of the conjunctiva," since it appeared to be unconnected with any other morbid condition, either local or general.

Von Graefe appears to have excluded, to his own satisfaction, trachoma, which is the disease that would naturally occur to anyone in connection with atrophy of the conjunctiva and trichiasis. But two cases have been met with in our out-patients, one four years, and the other two years ago, where almost obliterated conjunctival sacs and pannus were associated with cicatricial scarring on the inner aspect of the upper lids, and trichiasis. In both of these there was a history strongly suggestive of granular lids of long standing. But further observations are required as to the influence of trachoma and other morbid conditions, such as syphilis, in inducing atrophy of the conjunctiva.

ON MALIGNANT ENDOCARDITIS.

WITH CASES.

By FREDERICK TAYLOR, M.D.

I THINK it will be allowed that malignant or ulcerative endocarditis is still a subject of considerable interest, even though it has been made much more familiar of late years by the publication of many cases, and by the writings of different authors upon it. In February of last year I read before the South London District of the Metropolitan Counties Branch of the British Medical Association a short paper dealing with this disease; and much of that article I have incorporated in the present communication. But I have in the present paper added a considerable number of cases hitherto unpublished, of which some were under my own care, and are recorded in rather full detail; while others I am permitted to publish by my colleagues, and these are generally only briefly reported.

It is with some hesitation that I venture to put these cases before readers without the full bacteriological details, which are now so often required in the records of cases of this class; but my apology must be that several of them occurred in the pre-bacillary days, and the majority before our own bacteriological laboratory, presided over by Dr. Washbourn, was established. To a clinical teacher also it may be perhaps permitted to urge that the time has not yet come for laying aside entirely the familiar methods of clinical diagnosis, and for relying solely upon examination with the microscope and cultivation in nutrient media, but that differences between the pathological results of different infections may still be sought for and noted as a means to their recognition.

The disease we are now considering may be in a general way defined as a febrile process resulting from recent, often ulcerative, lesions of the endocardium, accompanied in many instances by evidences of embolic obstruction of vessels in various parts of the body. Illustrations of this will be found in most of the cases appended. I may shortly summarise one or two.

On July 19th, 1890, a little girl, æt. 11 (Case 3), was admitted under my care into Guy's Hospital, and she was subsequently, by the arrangements of our clinical wards, under Dr. Pye-Smith and Dr. Goodhart. She became progressively worse, and died on January 21st, 1891, six calendar months and two days after her admission. During the whole of that time, except the last six days of her life, she was uniformly and consistently febrile, the temperature being between 98° and 100° in the morning, and between 100° and 103° in the evening. On admission she was pallid, and she had a soft murmur of mitral regurgitation. These continued throughout her illness, and other indications of her disease appeared as time went on, namely, occasional rigors, early optic neuritis, and enlargement of the spleen.

Post mortem the chordæ tendineæ of the mitral valve were ruptured, the free ends were curled up, and the inner surface of the left auricle was covered with large fungating granulations. There were numerous infarcts in the kidneys and in the spleen; the latter organ was much enlarged; and there were old hæmorrhages on the surface of the brain. Micrococci were found in the cardiac vegetations by Dr. Pitt and Dr. Washbourn.

In Case 1, G. J. G—, a boy æt. 14, was admitted anæmic, febrile, with an enlarged spleen, cutaneous hæmorrhages, albuminuria, and a mitral murmur. Subsequently the pulses in the left radial, femoral, and tibial arteries became feeble or lost; he had double optic neuritis, slight hemiplegia, and paralysis of cranial nerves; and he died two months after admission. The inspection showed thickening of the mitral valve, with granulations and rupture of its chordæ, infarcts in the spleen and kidneys, and a large embolus in one popliteal artery.

Thus in each of these cases, and in those which are appended, the three important features of the developed disease were present, namely, (1) valvular disease as shown by the murmur; (2) continued pyrexia; and (3) embolic accidents in various parts of the body, with the results of the same.

I propose now to make some remarks on the disease, illustrating them where possible by the cases recorded.

NOMENCLATURE.

It is easy to find fault with a name, and the present complaint has not escaped the lot of other pathological and clinical disorders. It has been called *ulcerative endocarditis, diphtheritic endocarditis, infective endocarditis, septic endocarditis, fungating endocarditis, malignant endocarditis*; and it is contrasted in reference to each of these names with a "simple endocarditis." It is clear that some of these names are unsuitable. *Diphtheritic* is one of these. The disease has no special relation to diphtheria as we understand the term in England; there is no formation of a true membrane, and though organisms are found in the affected endocardium, they are not as a rule, though they may be exceptionally, those which are most distinctive of diphtheria. *Fungating* and *ulcerative* are both defective in being terms which apply only to the changes in the valve, and which do not carry with them an indication of the remote effects which follow in various parts of the body. *Septic* and *infective* are much more satisfactory, since they do really imply the share which sepsis or some process of infection has in the production of the results. It seems to me that infective is the better, since the term septic endocarditis, judging by analogy (*e. g.* septic pneumonia), rather suggests an endocarditis produced as a result of poisoning, while infective endocarditis seems to mean an endocarditis which itself brings about infection in other parts, and that is really the idea the name should convey. Advocates of septic might perhaps urge that septic things do, as a fact, produce sepsis elsewhere, and that granted an endocarditis was due to sepsis, other sepses must be ultimately the result.

Malignant endocarditis is, however, the term which has been most extensively adopted; but I feel it is not entirely good. In the case of tumours, malignant means persistent and rapid growth, recurrence after removal, reappearance in other parts of the body, and fatal tendency. In respect of only two of these characteristics is endocarditis malignant; it persists unaffected by treatment, and it has a fatal tendency. But nobody can remove it with the knife, and in respect of reappearance the endocarditis cannot develop in remote organs. It can only repeat itself within the limits of the endocardial surface. It is the micro-organisms which are malignant. Nor is the name distinctive as regards its fatal tendency; for endocarditis, simply chronic and constrictive as regards orifices, has an undoubtedly fatal tendency and serious remote effects—witness ordinary mitral stenosis, with pulmonary congestion, dropsy, and albuminuria,—and nobody would put these cases in the same category.

These considerations almost naturally suggest the question whether simple acute endocarditis—for instance, that of rheumatism or chorea is not itself the result of a micro-organism. Dr. Osler, in 1885, seemed to regard this view as a necessary outcome of the observations and researches at that time known, though, he says, "not perhaps completely demonstrated." The idea has been followed up by others since, but I believe it still remains an open question.

PATHOLOGY.

Whatever uncertainty or improbability there may be as to the bacterial origin of acute endocarditis in general, there is now none with regard to the malignant forms. Some time ago this condition of sepsis, the pyrexia, and other constitutional disturbances was more or less a mystery. It was attributed to the carriage in the blood of minute particles from the ulcerated surface of the diseased valves, and it was compared with pyæmia from lesions connected with the veins. Dr. Wilks called it *arterial pyæmia.* The uniform discovery of micro-organisms, whenever they have been looked for, explains, so far as the action of these bodies is understood, the special symptoms of these cases, or at any rate places the disease in the same category with other infective disorders.

But one of the most important and interesting facts with regard to the bacteriology of this disease is that there is no one organism peculiar to it. In an article on infectious endocarditis[1] it is pointed out that more than a dozen different micro-organisms had been found in different cases of the disease; that some of these have not been found in other disorders, and are so far peculiar to endocarditis; but that other organisms are those with which we are familiar in different well-defined infectious disorders. Those hitherto peculiar to endocarditis are the following:—A new bacillus discovered by Lion, *Bacillus endocarditis griseus* of Weichselbaum, *Bac. endoc. rugatus* of Weichselbaum, *Bac. endoc. capsulatus* of Weichselbaum, *Bac. immobilis fœtidus* of Fraenkel and Saenger, and a non-cultivable bacillus discovered by Weichselbaum. The other forms found in endocarditis are *Staphylococcus pyogenes albus* and *aureus*, *Staphylococcus pyogenes*, *Bac. pneumoniæ*, *Bac. tuberculosis*, and the bacillus of typhoid.[2]

These statements, if supported, are in accordance with the facts already known, that ulcerative and malignant endocarditis was liable to succeed every now and then to a preceding acute disease, such as pneumonia, upon which Dr. Osler[3] lays great stress, and with the fact attested by Dr. Goodhart[4] that chronic endocarditis was liable to take an ulcerative form in several cases in a hospital when some such disease as pyæmia, typhoid, or scarlatina was rife. The cases that I have analysed for the purposes of this paper afford comparatively little confirmation, whether clinical or pathological, of these facts; but that is largely because they were recorded without any reference to these considerations. I should say that I have before me the notes of fifty-three cases, of which seventeen have occurred under my own care over a long period. One is a case of which I saw the post-mortem by the kindness of Dr. Arnold Scott at the Fulham Infirmary, and the remaining thirty-five have occurred at Guy's Hospital under the care of my colleagues. Among these a large number are cases in which the patients have suffered mainly from cardiac valvular disease, upon which the ulcerative or fungating conditions have been grafted; and it would be a difficult inquiry, attended with uncertain results, to look back into the surroundings of each of these cases at the time of the fatal symptoms.

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[1] M. Rodais, 'L'Union médicale,' October 7th, 1890.

[2] See also Hamilton, 'Text-book of Pathology,' 1889, vol. i, p. 604.

[3] Gulstonian Lectures, 1886, 'Brit. Med. Journal.'

[4] 'Path. Transactions,' vol. xxxiii, 1882, p. 52.

But a few cases are of interest in reference to this question of infection. Most prominent are those cases in which the affected valves are on the right side of the heart, namely, the pulmonary and tricuspid valves. Of this there are eleven instances. In three cases (19, 22, 23) the pulmonary valves were alone diseased, and one of these patients had gonorrhœa, another vaginitis. The third was a man aged thirty-nine, in whom no antecedent lesion seems to have been present.

The pulmonary valve was diseased in association with the mitral in one case (25). The patient was an old man with enlarged suppurating prostate and suppuration of the prostatic veins. There were infarcts on both sides of the circulation—that is in the lung on the one hand, in the kidney on the other; there were also emboli in the cerebral vessels, with softening and suppuration of the liver and of the thyroid body.

Of five cases in which the tricuspid valves were involved, in two (21, 24) they were the only valves affected with ulcerative disease. One was a man who had sores on his hands; the other was a girl, æt. 17, in whom no source of infection could be traced.

In Case 52 the tricuspid was affected as well as the aortic and the mitral valves. The patient was a young man who had old valvular disease, with hypertrophy and dilatation of the left ventricle. He had an injury to the head, was trephined, and died of meningitis. At the post-mortem, made by Dr. Pitt, the sequence of events was doubtful. The meningitis may have been due to malignant endocarditis set up by the injury; the malignant endocarditis, on the other hand, may have been due to meningitis, itself the result of the injury, or even of the operation.

In another case (20) there was disease of the tricuspid valve and of the aortic at the same time. The patient had empyema and purulent pericarditis, and it must remain an open question whether these purulent collections provided the material by which the tricuspid valve was infected, or whether endocarditis, pleurisy, and pericarditis were all due to one common cause.

In the three remaining cases (11, 37, 48) the left side of the heart was chiefly implicated, and in one of these the patient was the subject of wide-spread malignant disease.

Tubercle was present in three cases, and perhaps in a fourth (Case 50). In Case 47 there was a caseous nodule with grey miliary tubercles in one upper lobe, and miliary tubercles in the lower lobe. Around the upper lesion was some pneumonic consolidation. Suppurative basal meningitis was also present, but no tubercles appear to have been found in connection with it. The affected valves were examined for micro-organisms, and micrococci were found, not bacilli, so that it is very doubtful if this can be regarded as a case of ulcerative endocarditis set up by the *Bacillus tuberculosis.* As will be seen presently, there is another group of cases into which it may perhaps fall.

In Case 15 there were caseous bronchial glands, and ulcers on the vocal cords and epiglottis. In Case 32 there were indurated lungs with old phthisis at one apex.

From the observations on the bacteriology of this subject already referred to, it appears that the different micro-organisms are apt to produce different pathological clinical results. Thus it is stated that some organisms invade the mitral by preference, others the aortic valve; that the condition of the vegetation or ulceration is dependent on the organism; that sometimes the infarcts suppurate, and sometimes they do not. Of one association I have seen a few instances; pneumonic infectious endocarditis is said to affect chiefly the aortic valves, and to be accompanied by suppurative meningitis. This was noted by Dr. Osler in his Gulstonian Lectures;[1] he says, speaking of meningitis, "In the majority of cases it occurred in connection with pneumonia." And the late Dr. Gulliver recorded cases arising in connection with pneumonia, in one of which there was early acute inflammation of the brain, not meningitis, and the endocarditis affected both valves, aortic and mitral; in the other case the aortic valves were alone affected, and the brain was healthy. In the present series there is a case (47), already referred to, in which there was basal suppurative meningitis, pneumonic consolidation surrounding caseous and miliary tubercle, and endocarditis of the mitral valve. In Case 12, under the care of the late Dr. Carrington, of which the post-mortem was made by Dr. Hale White, the aortic valves were alone diseased; there was purulent meningitis, chiefly of the bases, without tubercle; the patient had been acutely ill a week before admission, and clinically the lungs were regarded as being pneumonic. But post-mortem there seems to have been a little doubt as to whether the condition was not one of hæmorrhage rather than true hepatisation. In Dr. Scott's patient (Case 53) there was suppurative meningitis, but the valve affected was the mitral, and not the aortic, and the lung was found to be collapsed, and not recently pneumonic; but the patient's illness had commenced with pneumonia about six weeks previously.

Looking over the whole series of cases, there is still a great deal of uniformity in the main pathological conditions. Out of 52 cases in which the valves were found post mortem to be diseased, the mitral was affected alone in 23 cases, the aortic alone in 5, the mitral and aortic together in 13, the pulmonary alone in 3, the tricuspid alone in 2, the pulmonary with the mitral in 1, the tricuspid with the valves of the left side in 5 cases. (Of these 11 cases some mention has been already made.) Besides the lesions of the valves the most common pathological lesion was infarct in the spleen, which occurred 29 times in the 52 cases, and infarcts of the kidney were found 26 times. The infarcts were softened or suppurating in the kidney in Cases 25 and 27, and in the lungs in Case 26; and in these the condition was no doubt indistinguishable from pyæmia. The splenic infarct in Case 1 was soft, and those in Case 14 were "probably commencing to suppurate." In Case 46 were punctate abscesses, but none of the more common infarcts.

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[1] 'Brit. Med. Journal,' 1885, vol. i, p. 470.

Some implication of the brain is relatively common, and was found in nineteen cases, that is more than one third of the whole number. In nine cases hæmorrhage occurred, and the relation of this to the endocarditis, and its independence of the more common cause of cerebral hæmorrhage, is shown distinctly in most cases. In two of these, emboli were found in the cerebral vessels, and in three more, the hæmorrhage was directly due to the previous formation of an aneurism. The age of these patients is a most interesting point, for only one of the nine was over thirty years of age, the ages being 11, 12, 13, 14, 19, 19, 23, 25, and 31. Actual emboli of the cerebral vessels were found in six cases, and in one a wedge-shaped nodule on the surface of the brain, neither recent hæmorrhage nor veritable softening. In seven cases, partially overlapping those already given, there was recent softening or cicatricial evidence of the former softening. In five cases there was meningitis, of which two have been already mentioned (Cases 12, 47); the others are Cases 51, 52, and 53. As may be supposed, in a large number of these cases fits or paralysis appeared amongst the clinical symptoms.

Neither in regard to these pathological facts, nor in the consideration of the clinical symptoms, have I attempted to calculate percentages; for on the one hand the cases are but few, and on the other they have not been taken indiscriminately over a certain period of time, but some of them are in a manner selected.

SYMPTOMS.

The important clinical features of malignant endocarditis have been already referred to, namely, the cardiac murmurs, the pyrexia, and the evidences of embolic processes.

Murmurs.—A cardiac murmur is not, of course, in itself indicative of malignant endocarditis; it may depend on acute "simple" endocarditis, or chronic endocarditis, or on intraventricular or hæmic conditions independent of inflammatory changes. The existence of a murmur is chiefly of value in those cases of illness in which previous cardiac disease is not known to have been present. In that large proportion of cases where a chronic endocarditis takes on malignant properties, distinctive characters could only be looked for in the appearance of murmurs in connection with valves hitherto unaffected, or in a change of the quality or characters of the murmur already existing. I do not think the cases here recorded lend much support to the idea that, as between the aortic and mitral valves, the affection of either one would be distinctive either of an ulcerative lesion, or of the special form of infection causing it. I have already shown that pneumonia is not invariably associated with endocarditis of one particular valve. But it is otherwise with regard to the valves on the right side of the heart; they are so rarely the seat of acute endocarditis, that the implication of the pulmonary valves, or of the tricuspid in an acute febrile illness, with no preceding chronic cardiac disease, must be considered to point to the possibility of malignant endocarditis of the valve concerned. It is, however, not conclusive, as it is possible for a patient with anæmia and the associated pulmonary hæmic murmur to have febrile reaction from some independent cause sufficiently long to raise unfounded suspicions of ulcerative endocarditis. But a murmur, whether old or recent, may undergo variations in its character, or in its intensity, from the active changes that are taking place in the valve; from the varying amounts of fungating material; from ulcerative processes with perforation of the cusps of the valves; from the rupture of chordæ tendineæ, or detachment of flaps of the aortic or pulmonary valves. This has not been in my experience a very frequent feature; still, in my own seventeen cases, of which I have the most full details, I have it noted certainly in four cases, and doubtfully in two others. In these four cases the murmur became louder, or was heard over a larger area, or it acquired a musical or whistling character. A musical quality is undoubtedly liable to be the result of the alterations of valve structure which the ulceration leads to, especially perforations of valves, detached flaps or strips, and ruptured chordæ; such pieces apparently vibrating in the blood-current with sufficient regularity to produce musical sounds. Hence to a certain extent a musical murmur may be suggestive, though by no means conclusive, of the disease in question. From the notes before me I can only find a record of musical murmur in six cases (Nos. 2, 3, 8, 9, 29, 42); and of these the aortic valve was perforated in two instances, the mitral chordæ ruptured in one, while in the other three cases there were vegetations only. On the other hand, there are cases where the chordæ are ruptured without the murmur having acquired a musical quality.

In reference to diagnosis, attention must be called to the occasional absence of murmur. Of course, the fact may be doubted; it may be said that if it was not heard, it was because the observers were careless, or did not examine with sufficient frequency. In nine cases out of the fifty-three I find no murmur is recorded. In Case 24 "at no time was any murmur heard over the heart;" in Case 5, on the eleventh day of his illness, the day before his death, no bruit was audible; but according to the history he had had rheumatic fever, and had been told by a doctor he had heart disease; in Case 48 the heart sounds were normal on admission, but the patient, after removal of a tumour of the eyeball, died of wide-spread malignant disease, and it is possible that the cardiac affection may have come insidiously late in her illness; in Case 20 there were complicating conditions, namely, pericarditis and empyema, with consequent surgical operation; and it is quite possible that the endocarditis was of late origin. Of two other cases I have not been able to procure the clinical notes.

With the murmur we may note the condition of the heart, which will be dependent to a certain extent upon the fact whether present trouble has been grafted upon old disease, or upon practically healthy valves. In a case coming before one for the first time, the occurrence of irregularity or displacement of the apex will prove or suggest an old disease, which may have served as material for the invasion of the micro-organism.

Pyrexia.—This is as constant a symptom in malignant endocarditis as the presence of a murmur. It occurred in thirty-eight out of the fifty-three cases; and there can be little doubt it was present in many more, of which I have not got the full clinical notes, or of which the temperature charts have been lost. Pyrexia may indeed be overlooked in those cases, of which a good many occur, where the ulcerative changes supervene upon chronic valvular disease; the patients are under treatment for dropsy, cyanosis, albuminuria, the results of imperfect compensation, when the more active and serious lesions of the valves are grafted on the old disease, and rapidly, it may be, prove fatal. There is another group of cases, in which the patient has first come under observation with a fit or paralysis, when the local cerebral conditions may disturb the conditions of temperature. But in those instances where either the valvular disease is entirely recent, or if old has not yet disturbed compensation, the fever becomes one of the most prominent features of the illness. The possible variations of a temperature chart are so few in comparison with the multitude of known diseases, and the variations of temperature in the same disease are so numerous, that it is by no means always safe to associate particular features of temperature records with special diseases. What occurs in malignant endocarditis is, as a rule, the remittent or intermittent course which is characteristic of so many septic conditions,—typhoid fever, tuberculosis, phthisis, and prolonged suppuration under certain conditions—a high evening temperature, and a morning fall to a point near or not far above the normal.

Dr. Osler laid much stress on the irregularity of the temperature. I am not prepared at present to agree with him. The temperature of some of my cases has been remarkably uniform and regular over periods of weeks and months. Cases 1, 3, and 7 were of this kind. I do not mean that irregularity of temperature does not occur, but that it cannot be considered characteristic. Where there are complications, such as those of pneumonia, or meningitis, or pleurisy, it is likely that the temperature will be constantly higher, as in

Case 47 with basal meningitis. And it is intelligible that rigors should disturb the even course of the temperature, though such disturbance may be but slight, and the rigors may be very infrequent. Rigors associated with pyrexia were observed in twelve cases, and probably occurred in some other instances. Perspirations are noted only in eight cases; these also may have been in some others overlooked. There was rigor or perspiration in all the cases of lesion of the pulmonary valve, but, as the figures show, the cases of left side disease were not exempt.

Obstruction of vessels.—This is perhaps one of the most interesting features of malignant endocarditis, and it is one to which the attention should be at once turned when a case is suspected. This obstruction takes place, as we know, by the impaction of emboli, or particles carried off from the diseased valves.

Such embolisms may occur in almost any part of the body, but some parts are more liable than others, such as the spleen, kidneys, and brain.

The evidences of embolism may be classified into the following six groups:

A. *Absence of pulsation in accessible vessels,* such as the radial, tibials, or brachials. One pulse is lost, or no pulse can be felt in a tibial artery. This is, however, not very common. In Case 1 we have the note, "Left radial pulse feebler; femorals scarcely detected; tibials not to be felt." This was a month before his death. He afterwards had hemiplegia and convulsions from obstruction of the middle cerebral artery. In Case 18 the left posterior tibial and dorsalis pedis were pulseless, and embolism of the left common iliac artery was found post mortem.

B. *Loss of function* from obstruction of vessels not readily or not at all accessible; for instance, hemiplegia from embolism of a middle cerebral, which is much more frequent than pulselessness in the limbs.

The subject of cerebral embolism has been already referred to; as a clinical fact paralysis was noted eight times (Cases 1, 8, 12, 28, 42, 46, 49, 51), and the paralysis was in all cases but one hemiplegia, but in one instance it was due to meningitis, and not to arterial obstruction. Fits or convulsions were present in eight cases, and some of these have a relation to embolic processes somewhat different, or at any rate a little more remote. For in five of these cases the fits were associated with cerebral hæmorrhage; and in these five cases it has been already shown that embolism or aneurism was present. In two other cases the brain was found to be healthy, and in the last no post-mortem was made. The total number in which either fit or paralysis occurred and was related to embolism was twelve.

Blindness or loss of vision from embolism of the arteria centralis retinæ may be included in this category.

C. *A function may be disturbed*, as in the case of albuminuria, which is often noted; it may be due to embolism and infarct in the kidney, or to a form of nephritis which is often seen in malignant endocarditis. The last would come under another heading.

D. *Obvious infarcts*, as the term is commonly employed. These may be reasonably assumed to exist when the spleen is considerably enlarged. It has already been shown how exceedingly common is an infarct of that organ under the circumstances; and if with fever and murmur the spleen progressively enlarges, the probability of infarcts is great. In Case 1 the spleen at one time reached nearly to the umbilicus, and though it subsequently diminished a little, there could be no doubt of its relation to the endocarditic process. At the post-mortem the spleen was described as "practically one large soft yellow infarct."

E. *Hæmorrhages.*—Hæmorrhage into the substance of an organ is well known to be the result of embolism; an intense congestion takes place within the area of distribution of the obstructed artery, and the minuter vessels rupture, and the blood either soaks into the tissue, or if it finds exit, gets away on to a free surface. Hence many, if not all hæmorrhages under these circumstances may be looked on as results of embolism. I should suppose, however, that some of the smaller hæmorrhages may perhaps be regarded as holding the same relation to the bacterial virus of endocarditis as those of smallpox and typhus do to the virus of these diseases—that is, that they are toxic in origin. And it seems to me now to be an inquiry worth instituting, whether particular groups of malignant endocarditis are more liable to minute hæmorrhages than others.

Dr. Sidney Martin, in his interesting Gulstonian Lectures,[1] shows that in a case of infective endocardis which he investigated the valves presented a micrococcus growing in clusters; and in the blood and spleen he found albumoses and an acid body, the results of the growth of the coccus in the valves. And he points out that he has shown that the jequirity seed contains "two poisonous proteids, a globulin and an albumose, both of which produce in very small doses a general disease, two of the features of which are the production of ecchymoses and the occurrence of sanguineous diarrhœa."

The hæmorrhages which result from embolic processes in the viscera, such as the brain, spleen, lungs, kidneys, have been already noticed. Petechiæ under the skin occurred in eight cases. Retinal hæmorrhages were seen in Cases 6, 7, 17, and 49, and I have observed the same in a case which I saw in consultation with Dr. Sydney Smyth of Brockley. Hæmoptysis, hæmaturia, and epistaxis are less frequent occurrences.

F. *Inflammatory lesions.*—Whether these should be considered as due to minute embolism, or as septic in a more general way—that is to say, whether if septic the organisms are carried by particles detached from the diseased valves, or whether the organisms are simply diffused through the blood, and hence in particular organs set up inflammatory lesions, or whether the chemical products of bacteria are influential as a cause of the lesions—may be open to question. Dr. S. Martin's researches on the albumoses of diphtheria[2] support the last explanation, but it may be remembered that micro-organisms (streptococci) have been found during life in the blood of patients affected with malignant endocarditis.

The inflammatory conditions which are most frequent in association with malignant endocarditis are nephritis, optic neuritis, and suppurative meningitis.

The lesions of the kidneys are of very great interest in this disease. It has been already shown how frequent are infarcts of this organ, but the inflammatory lesions are independent of these, though the two conditions may coexist.

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[1] 'Brit. Med. Journal,' vol. i, 1892, pp. 756, 758.

[2] Loc cit., p. 699.

In quite one third of the cases nephritis in one form or another was found. Some of these may have been of old standing, such as the fibroid glomeruli in Case 17, and the cirrhosis and wasted glomeruli in Case 44. In some other instances also the lesion was probably not connected with the endocarditis directly. In Cases 24 and 25 there was suppurative nephritis,—in the one "pin-points of suppuration, as if from infarction," in the other "pyæmic infarcts." In Case 27 there was old ascending nephritis.

In all three cases the right side of the heart was involved, but the renal lesions in two of the cases (25, 27) were undoubtedly related to the condition of the lower urinary passages—stricture and large prostate.

Fatty kidneys with old cysts were present in Case 10, an elderly man with aneurism of the profunda femoris, and subacute nephritis with fatty changes in Cases 31 and 32; both had suffered from cardiac disease for some time. In Case 4 there was fatty degeneration of the epithelium, and a considerable amount of interstitial change together with lardaceous degeneration.

In several other cases the change would seem to be much more directly related to the disease we are considering. Thus in Cases 2, 3, 5, 17, 19, 29, 41, and 49 the kidneys were mottled, and in 45 they were rather swollen, and in Cases 1, 7, and 35 there was clearly an acute inflammation. A very interesting state of the kidneys, with which I have been familiar for some time, and which has been specially noted several times by Dr. Pitt and Dr. Perry in the post-mortem examination of these cases, deserves particular mention; namely, a uniform appearance of ecchymosis in minute points or petechiæ, dependent upon hæmorrhages in the glomeruli.

This is sometimes present with acute inflammation or mottling, but it may be doubted whether it is to be regarded as an inflammatory condition, or merely one of ecchymosis or hæmorrhage such as occurs in other parts of the body, for instance, the skin and retina. It was present in ten cases, and is described as follows:—Numerous punctiform hæmorrhages (Case 2); some glomeruli marked out with petechiæ (Case 3); on section, numerous punctiform red spots in the cortex (Case 27); a few minute punctiform ecchymoses in the cortex (Case 28); the cortex marked by minute extravasations, on section the same petechiæ with linear extravasations (Case 14); surface and cortex riddled with minute petechial spots, which occupied the Malpighian corpuscles, and stood out as red spots on a mottled stone-coloured ground (Case 36). In Case 35 Dr. Pitt, who made the autopsy, has given a detailed account of the state of the kidneys. "The capsule peeled readily. The surface was densely covered by minute petechiæ on the substance of the kidney, with mottling with minute spots; and the same condition was apparent throughout on section. The peripheral parts of the medulla were congested. This condition I looked upon as an acute nephritis, but Dr. Perry has previously noted its association with malignant endocarditis; there were no medium-sized petechiæ on the surface of the pelvis or of the kidney, such as are often seen with abdominal congestion, blood-poisoning, &c. Microscopically the kidneys show universal glomerular nephritis. The glomeruli show lunulæ of fusiform cells, extensive multiplication of nuclei of capsules and capillaries, so that the capsules are crammed with cells. There is also a general, but not extensive, cell-infiltration. Many convoluted tubes are dilated. There are a few collections of small cells, but none like abscesses or infarcts. The epithelium of the tubes is not bad. The glomerular changes are, many of them, old, but the naked-eye appearance points to an acute condition superadded." Cases 16, 29, 41, are other cases in which ecchymoses were noted.

I have already referred to suppurative meningitis, and its relation to pneumonia.

Optic neuritis, the other form of inflammation which is sometimes seen, may undoubtedly be the result of meningitis when this is present, as perhaps happened in Case 47; but it also most certainly occurs in malignant endocarditis without cerebral complications, and I am inclined to regard it as useful in confirming a diagnosis in some instances. In Case 3 there was undoubtedly early neuritis, which I observed in July, and which was still visible, in the form of moderate blurring of the edges of the two discs in October. But when I saw her shortly before her death it was not recognisable as neuritis, and the appearance only allowed me to say that possibly such a thing had been. In other cases it has been more marked and persistent. The proportion of cases in which it occurs I cannot speak to. I have searched through some reports without finding mention of it, though I cannot always say that it has been looked for; I have only investigated the point the last few years. Besides Cases 3 and 47, it was present in Cases 1, 4, 16, and 49. Its diagnostic value must, however, be qualified. For instance, enteric fever is occasionally the cause of optic neuritis, and it is just the disease which requires to be diagnosed from malignant endocarditis.

In regard to meningitis, if the discovery of optic neuritis leads to the diagnosis of meningitis, this may be a step towards the recognition of malignant endocarditis as a cause or associated condition.

In Case 53, associated with meningitis, there was a suppurative inflammation and destruction of one eyeball (panophthalmitis).

There is, lastly, as an interesting feature in malignant endocarditis, a symptom to which I am inclined to attach a good deal of value, and that is *anæmia.* Pallor or anæmia is noted in 24 instances out of the 50, and often it has been very pronounced. Its connection with malignant endocarditis must, however, be carefully scrutinised. For instance, it is well known that aortic regurgitation causes anæmia, and in a certain proportion of cases the septic process is grafted on this disease. Similarly mitral regurgitation in children is frequently characterised by anæmia, and hence in Case 3 the anæmia did not at first impress itself upon me as a symptom having very special significance. The anæmia is often especially marked where the valves on the right side of the heart are involved.

In addition to the symptoms and pathological conditions which have been already recorded, the following will be found to have occurred in the cases here given:—albuminuria in 24 cases; delirium in 10 cases; vomiting in 7 cases; abdominal pain in 4 cases; erythema in 3 cases; orchitis in 1 case. Pneumonia was found in 12 cases; cardiac induration of the lungs in 12 cases; nutmeg liver, in 9 cases; pericarditis, old or recent, in 9 cases; ulceration of the colon, peritonitis, and lardaceous disease, each in one case.

DIAGNOSIS.

The diagnosis presents itself under two aspects, that is according as the patient is or is not suffering from failing compensation of the heart. It has been already remarked that ulcerative or "malignant" changes may graft themselves upon old disease. If the patient is actually under treatment for this cardiac failure, for irregularity of heart, dyspnœa, dropsy, and the other familiar symptoms, the additional features which are due to the infective element may not so readily be recognised; that is to say, the disease may be overlooked, because there seems already to be enough in the case to account for what is happening, and even for a fatal result.

Pyrexia, variability in the character of the murmur, embolism of the cerebral arteries or vessels of the limbs, enlargement of the spleen, hæmorrhages, or acute inflammations would be the conditions likely to awake suspicion.

On the other hand, the disease may be confounded with other complaints in those cases in which, whether the patient is known to have a cardiac murmur or not, he is not at the time suffering from failing compensation, and the pyrexia (and possibly some other symptom) comes upon himself and his medical attendant as the first beginning of the illness.

A disease for which malignant endocarditis may be and often is mistaken is typhoid fever. The subtleties of typhoid fever—its insidiousness, its frequent deficiency in characteristic symptoms apart from pyrexia, the absence of spots for many days, the absence of diarrhœa for many days—are well known. I was asked at the bedside a short time ago, "Do not most cases of typhoid fever have constipation?" and I had to draft the answer rather carefully. Here also we have an insidious disease with pyrexia, enlarged spleen it may be, and little else, unless the heart is examined. And if the heart is found to present a murmur, surely patients with old endocarditis are not exempt from typhoid fever. A murmur or an irregular pulse should put us on our guard, and then the following should be watched for:—rigors, which are rare in typhoid; petechial spots; absence of pulse in any accessible arteries; anæmia.

On the whole the fever is more uniform, not presenting the three weeks' course of typhoid; and the patient's mental condition is less disturbed, more tolerant of the pyrexia than it seems to be in typhoid. The patient may amuse himself by reading or otherwise all through, whereas the typhoid patient is more often dull, apathetic, and self-contained.

Again, I may remark, I do not believe that reliance can be placed upon the irregularity of the fever. I should be inclined in long-standing cases to go quite the other way. I certainly think the pyrexia is more uniform over a series of weeks than that of phthisis.

In reference to the diarrhœa of typhoid, it is important to notice that in at least eleven cases diarrhœa was present, and that in six of these (Cases 1, 3, 5, 6, 7, 24) the motions were loose, yellow, offensive, and in two or three instances were described as typically typhoid.

Pyæmia, ague, and general tuberculosis are other diseases from which the disorder may have to be diagnosed. With regard to the first the resemblance may obviously be very close, the two conditions may be present together—are they not the same to a certain extent? The organisms of pyæmia are some of the agents of malignant endocarditis. In some such cases, as already shown, the valves of the right side of the heart are affected. But what used to be the more common form of pyæmia would have been distinguished, I think, by more persistent and frequent rigors, and by the pulmonary lesions, abscesses in the lungs, and pleurisy, which are not at all common in malignant endocarditis.

I may here perhaps allude to a case which I have appended at the end of this paper (Case 54), and in which the diagnosis of ulcerative endocarditis was made during life. He had aortic disease, a temperature oscillating between 101° and 105°, and albuminuria. There was no acute endocarditis, but there was ulceration of the aorta above the valves, by which it communicated with two sacs in the ventricular wall, and one of these was practically an abscess with grumous contents.

From ague it differs—at least the more common cases—in the less pronounced regularity of the rigors, and the want of a healthy intervening period of apyrexia.

PROGNOSIS.

This, as is well known, is very bad. Nearly every case of pronounced ulcerative endocarditis is fatal; but it appears not to be absolutely hopeless. Dr. Pye-Smith, in a lecture on the "Prognosis and Treatment of Heart Disease,"[1] records a case of recovery, and on the occasion of reading a short paper on this subject before the Brixton Medical Society, I was told by Mr. Galton of a similar case under his care. Dr. Hale White informs me he saw a case with Dr. F. R. Humphreys, of which the features were cardiac dilatation with mitral murmur, rigors, and oscillating temperature. He regarded it as malignant endocarditis; but the patient recovered. It seems likely that there are other cases of this kind occurring from time to time.

One might indeed ask, why should it not sometimes recover? It is only one of a number of diseases attributed to the action of micro-organisms, and of these there is not one that is invariably fatal, unless it be hydrophobia. It is perhaps all a question of the quantity of the micro-organism introduced, or of phagocytes, or of whatever else is a factor in the protection of the body and the exhaustion of the disease. But if prognosis as to termination is on the whole more certain than we wish, the prognosis as to duration is very unsafe. The length of time during which a case will persist with not very much change, except perhaps progressive weakening, should be borne in mind. Case 1 lasted three or four months, Cases 2 and 3 at least six months; and other longer cases are on record. On the other hand, a few days or weeks suffice to carry off the patient in some instances.

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[1] 'Brit. Med. Journ.,' 1890, vol. ii, p. 1422.

TREATMENT.

Our present means of actively dealing with the complaint are entirely inadequate, we might almost say none at all, and probably all our hopes must be centred in the bacteriologist, in Pasteur and Koch and their disciples, present and future. We may, of course, follow the symptomatic indications. Temperature may be beaten down with antipyretics, but to very little purpose. If the temperature does rise to 103° or 104°, it will almost certainly come down within a few hours, and if you beat it down it will go up again in a few hours. The persistent use of these drugs will probably only result in delaying the late afternoon rise till the evening or midnight. Sweating may be checked by a drop of Liquor Atropinæ Sulphatis at night or other suitable time. Pain may be relieved by opium, and general support may be given by what must still be called tonics, such as iron, quinine, nux vomica, and good food. But I should not give quinine as an antipyretic, nor as an antiperiodic (if there are such things). Nor have I hitherto been sanguine as to any permanent good, though I have used them, from sulphocarbolates or benzoates employed as antiseptics. There seems to me no reason why we should expect to get better results from antiseptics in this disease than we do in phthisis, diphtheria, scarlatina, and a number of other infective disorders. Perhaps the doses have not been large enough, and at present it must be allowed that this offers the only chance of influencing the disease apart from the general support mentioned above.

The cases which follow have been roughly classified into five groups:

Group I.—Eighteen cases in which the symptoms were general or typhoid in their character; that is to say, pyrexia was the prevailing feature, and the patients did not suffer strikingly from cardiac failure.

Group II.—Nine cases in which the symptoms or conditions were approximated more closely to those of pyæmia; in seven of these the right side of the heart was involved.

Group III.—Thirteen cases in which cardiac symptoms were prominent, the patient often having dropsy, large liver, albuminuria, or other indications of deficient compensation.

Group IV.—Six cases in which cerebral embolism or aneurism complicated the cardiac disease.

Group V.—Seven cases, including some rather special instances in regard to course or complications. In this group are four of the cases with meningitis, one of multiple cancer, one of disease of the hip, apparently tuberculous, and one of aneurisms on both vertebral arteries.

The classification is necessarily imperfect, as all such pathological or clinical groupings are known to be; it is rather an attempt at a convenient arrangement in order.

GROUP I.

CASE 1. *Malignant endocarditis of mitral valve; pyrexia; anæmia; optic neuritis; albuminuria; hemiplegia; obstruction of vessels; infarcts in spleen and kidney.*—G. J. G—, æt. 14, was admitted under my care, March 17th, 1886. He has never had rheumatic fever. Twelve months ago he fell down some stone steps and struck his occiput. He was picked up unconscious, brought to Guy's Hospital, and remained comatose three days. He returned home. There was no bleeding from the ears. He was well in six weeks, but soon after was troubled with vertigo. He never fell in these attacks until six weeks ago, when he fell into the road. Lately he has complained of pains in the left leg and headache. He has lost appetite lately, and has had chiefly milk and beef-tea.

On admission.—He is very anæmic, and lies on his side with his legs drawn up, and arms fixed in between them. He complains of sharp pain over the stomach, and has much tenderness there. He is very drowsy, but answers readily questions put to him. The pupils are equal, and react to light and accommodation. There are petechiæ over the greater part of the body; the legs alone are free. The cardiac impulse is in the fourth space; it is internal to the nipple, and rather diffused. There is a loud blowing systolic murmur at the apex, extending to the base, where it becomes less distinct; it is not traceable into the axilla. The second sound is accentuated.

The lungs are normal, except that air enters the bases badly. The spleen is slightly enlarged, and extremely tender, so that the patient cannot sleep on the left side. There is also some hardness and tenderness in the left groin, thought to be due to lymphatic glands. The left optic disc is rather full and blurred on the inner side; the vessels tortuous; no hæmorrhages.

On the 19th, the urine was found to contain a small quantity of albumen. The patient complained during these first few days of severe pain on the left side of the abdomen, and occasionally vomited. His temperature was constantly above the normal, being from 101° to 103° in the evening, and 99° or 100° each morning.

On March 24th the bruit had a whistling character.

On the 29th the spleen was found projecting two inches below the costal margin. Dr. Goodhart, who had charge of the case for a time, noted that the second sound over the pulmonary artery was very accentuated, and had a sort of systolic purr accompanying it, suggesting vegetations on the aortic valves.

On the 30th he was complaining of pain in the knee. The joint was not swollen, nor did it grate.

On April 2nd the petechiæ were fading, and some subconjunctival hæmorrhages had cleared up.

3rd.—Pain in both knees.

5th.—Much pain in both knees; puffiness of the popliteal spaces, and pain on pressure; veins prominent.

8th.—Pain in the left elbow.

12th.—Left radial pulse feebler; femorals scarcely detected; tibials not to be felt; epistaxis.

13th.—Headache and vomiting; anæmia noted.

18th.—Does not understand readily what is said to him.

21st.—"Drawing" pains in the head; calls things by wrong names; hearing defective.

22nd.—Double optic neuritis.

24th.—Less drowsy; more rational.

30th.—Slept well, but now screaming with pain in the head, throwing himself about, often putting his hand to the back of his head, but will not say where the pain is. At another time in the day had rigidity and paralysis of the left arm and weakness of the left leg. Both eyelids were dropped and the eyes turned to the right; occasional movements of nystagmus; pupils dilated; slight left frontal paresis.

May 2nd.—Continued slight left hemiplegia, but can speak.

3rd.—Paralysis of right internal and inferior recti.

10th.—Can move the left arm and leg now fairly well.

11th.—Irregular action of the heart; left radial pulse cannot be felt.

14th.—Face drawn to the left; left side much wrinkled, but weakness mainly concerns lower half. After this articulation became impaired.

On May 22nd, at 5 a.m., he was stertorous, with a rigid left arm. Shortly after this he had convulsions, chiefly on the right side, then in all four limbs. Cheyne-Stokes breathing came on, and he died at 2.30 p.m.

Autopsy, May 24th.—The *pericardium* was healthy.

The *heart* weighed 6 oz.; all the cavities were in diastole and empty of clot; the cavity of the left ventricle was, however, small. The posterior cusp of the mitral valve was much thickened, as well as its chordæ. Several of these had snapped and were curiously twisted up. There were numerous granulations on the cusp.

Lungs normal, neither brown nor indurated.

Liver 37 oz., healthy.

Spleen 5 oz., practically one large soft yellow infarct.

Kidneys 7 oz., acutely inflamed, and containing several infarcts.

Brain weighed 52 oz. The left middle cerebral and the right anterior cerebral arteries were blocked by emboli; a careful search revealed no others. The amount of cerebral softening was very extensive; it was chiefly situated on the right side, and the extent would probably be best indicated by saying that all parts above and in front of the posterior branch of the Sylvian fissure were involved. The right optic thalamus was shelled out, but the corpus striatum was intact. In the centre of the softening, and abutting on the surface just above the Sylvian fissure, were three ounces of recently extravasated blood. There was another large patch of softening, involving nearly the whole of the left frontal lobe. The hæmorrhage on each side had extended into the lateral ventricles; but it appeared certain that there was no communication between them through the foramen of Munro.

Limbs.—An embolus was found impacted at the bifurcation of the right popliteal artery. The left brachial, axillary, and radial arteries were found free from emboli.

CASE 2. *Aortic endocarditis, extending to mitral valve; pyrexia; rigors; albuminuria; infarcts in spleen.*—Samuel B—, æt. 34, was admitted into my ward, under the late Dr. Wooldridge, on January 18th, 1888, and subsequently came under my care.

He had jaundice seven years ago, was ill six or seven weeks, without pain, but was not ill enough to go to bed.

Has never had scarlet fever or rheumatic fever. Has had chancres and gonorrhœa, but no further evidence of syphilis.

For some years has had pain in his back on walking much. Last June he became weak and tremulous, so that he could not do his work properly. Two or three weeks later he had cough; then he went to Brighton for a week, and while there had attacks of sudden blindness in the right eye, lasting two or three minutes. Once after sitting in the sun he could not speak, and on another occasion could not open or move his jaws. Two or three months later he was able to go to work again. Three weeks ago he had cough, weakness, fluttering sensation in the chest, and was tremulous.

On admission.—He is pale, with puffy face, swollen eyelids, and slight œdema of the feet. Slight cough, no dyspnœa, muco-purulent sputum with slight red tinge; a few râles in the right side of the chest. Pulse 108, regular, long, jerky. Precordial dulness reaches the third space above and the right border of sternum. Impulse in the fifth space, half an inch below and a quarter of an inch outside the nipple. Systolic bruit at the apex with reduplicated second sound; systolic and diastolic murmurs over both aortic and pulmonary areas at the base. The liver is felt just below the edge of the ribs; the spleen is not felt, but there is tenderness over it. Urine high coloured, sp. gr. 1028, contains a trace of albumen.

From the date of his admission the illness lasted six months, and he died on July 14th. During the whole of this time the temperature was febrile. It was, as a rule, between 98° and 99° in the morning, and about 101° in the evening. Shortly after admission, as already stated, it was above this level, and again the first few days of February. On the 4th, 5th, and 6th of March it rose, and on the 5th the morning temperature was 101°, the evening 104·8°. From May 10th to May 14th inclusive, he had five grains of antifebrin at 5 p.m. daily, with the apparent effect of limiting the evening rise, especially on May 13th, 14th, and 15th. From the 16th to the 23rd inclusive, three grains were given daily, with no such marked result; the last four days the temperature was about the same as it had previously been.

On January 23rd he had a rigor, followed by profuse sweating, and cold sweats were noted on February 29th, on March 24th, and on some other occasions. When first admitted he was delirious, and this condition lasted some days. Later on he complained of odd sensations, flutterings in the chest and abdomen, and showed a nervous and peculiar manner. A fortnight before his death, he became again delirious, with delusions, and on one occasion managed to get at some methylated spirit and drank it; this necessitated the use of the stomach-pump, and he appeared to be none the worse for it.

The *cardiac murmurs* remained on the whole much as at first: an aortic to and fro, a pulmonary systolic with clear second sound, the diastolic aortic heard loudly down the sternum; at the apex a double first sound with single second and a soft systolic murmur.

On March 31st the apical bruit was more musical, and its musical quality was noted on April 14th and 17th. But on April 30th I observed that this feature increased as the bruit was traced upwards, and that probably the musical note was of aortic origin. On May 12th the apex bruit was simply blowing and not musical. About this time also the murmurs became more audible behind, and a systolic thrill was felt in the second right intercostal space.

The *pulse-rate* varied from 90 to 106, but during the last fortnight was more constantly over 100. Pulse tracings, taken on March 6th and March 27th, showed the characters of aortic regurgitation.

There was no prominent trouble about the *lungs;* on admission he was spitting muco-pus with a little blood, and this was present for about a fortnight. Physical examination never revealed more than a little dulness and râles at the bases.

The *liver* enlarged slightly during his illness, so as to be felt two fingers' breadth below the ribs, and it was nearly always tender.

The *splenic* dulness was increased on March 13th, though the organ could not be felt; but it became palpable on March 26th, and was felt on several occasions afterwards. On July 9th, however, it is stated that it could not be felt. He complained occasionally of flatulence, but otherwise the alimentary system presented no special features.

The *urine* contained a trace of albumen on admission. Albumen was again found on March 6th in small quantity, and continued to the end.

I examined the optic discs on March 1st; they were rather red, but there was no optic neuritis. On March 19th also the discs were normal. There is no note of any later examination.

He was described as pale when admitted, but anæmia is especially noted on April 6th, and again on May 3rd and May 28th.

The only other conditions that may be noted were occasional headache, pain in the right eye, and complaint of inability to see with that eye (March 7), slight indications of facial paralysis (March 9), which were not again observed, and short attacks of giddiness (April 5th).

His treatment was at first digitalis and quinine with expectorants. On February 18th he was ordered salicylate of sodium. Antifebrin was given in five-grain doses for a few days, as already stated. On June 11th quinine and iron, and on June 14th Liquor Hydrargyri Perchloridi and Tinctura Cinchonæ Co. were ordered.

Autopsy.—Recent pleurisy over the left lower lobe, with typical red hepatisation of whole of this lobe; the remainder of the lungs was healthy, though rather tough. The bronchial glands were acutely swollen.

The *heart* weighed 19 oz. Left ventricle hypertrophied and dilated. Extensive ulcerative endocarditis of aortic orifice, which was considerably obstructed by masses of vegetations, while the valve was incompetent from a large perforation in one valve the size of a shilling. The endocarditis had spread by contact to the aortic cusp of the mitral. The mitral orifice was rather dilated. There were one or two vegetations on the chordæ tendineæ of the mitral valve, but not near its edge. The right heart and left auricle were normal. Section of the heart showed extreme fibroid changes in places, and muscular tissue replaced by delicate fibrous tissue. Arterial capillaries, running transversely among the muscular bundles, had rather thick walls, but were everywhere patent.

The *liver* weighed 80 oz., was nutmeggy on section, and rather tough.

The *spleen* weighed 29½ oz.; it showed numerous infarcts of various dates; some quite recent, and others decolorising; and one large scar, containing a yellow centre, represented the oldest stage.

Kidneys 18½ oz.; large and injected in a mottled manner, and containing numerous punctiform hæmorrhages on the surface of the cortex. The capsules stripped off easily.

Case 3. *Mitral endocarditis; pyrexia; optic neuritis; anæmia; infarcts in spleen and kidneys.*—Minnie G—, æt. 11, was admitted under my care on July 19th, 1890.

She is one of a family of nine, three of whom are dead, and the others are not very healthy. Her mother has had rheumatism, and suffers from her heart.

The child was healthy, with the exception of "remitting fever," till eighteen months ago, when she began to have dyspnœa, palpitation, and attacks of faintness. She then wasted and lost appetite. Some months ago she began to limp, and complained of pain in the right hip-joint, and five weeks ago she was taken into the Evelina Hospital, and was in three weeks. She then went home, and was kept in bed with a weight attached to the leg for ten days.

On admission.—She is fairly well nourished, fair complexion, pale, but the lips and gums of good colour. The chest is normal, except at the right apex, where the entry of air in front is not good, and behind the expiratory sound is prolonged. The pulse is regular, 106, rather small. The heart's dulness is normal; the impulse in the fourth space rather diffused, most marked under the nipple, rather forcible. There is a systolic bruit, loudest at the apex, but audible in the axilla and behind, and indeed over the greater part of the chest.

The temperature on admission was 102·4°, the following evening it was 101°, and for the next three weeks it ranged between 98·4° in the morning and 101·4° in the evening, being for the most part between 99° and 101°. She was ordered small doses of Liq. Strychninæ and Tr. Digitalis.

Towards the end of August she complained of pain in the umbilicus, and on examination some tenderness of the left iliac region and pain about the left hip-joint were noticed.

August 22nd.—The systolic apex bruit is quite musical and high-pitched.

September 8th.—Some tenderness over the spleen.

15th.—During the last week she has had two shivering fits.

17th.—A rigor again last night. She seems worse this morning; the tongue is furred, she has headache, and some pain in the splenic region. The urine is acid, free from albumen.

21st.—The spleen can be just felt below the costal margin.

27th.—There are indications of optic neuritis; the edge of the right disc is indistinct, of a violet colour, and the disc is somewhat hazy. The veins outside the disc are more dilated than those on the disc. The left disc is a little hazy.

October 4th.—The child is now decidedly anæmic. She again complains of pain in the left hip. The apex murmur is loud and musical, and the second sound is accentuated.

6th.—Dr. Washbourn reported that he had made some cultivations from the blood of this patient, and that he had obtained a growth of *Streptococcus pyogenes.*

The temperature varied now between 98° in the morning and .102° or 103° in the evening. She had rigors on the 7th, 9th, and 10th, but these did not appear to influence the temperature. On October 16th the right hand was numb, tingling, and weak; but it was better the next day. Antipyrin and antifebrin were given in small doses at this time, but caused sickness, and were therefore discontinued. Abdominal pain was troublesome, and was treated by hot fomentations and poultices.

27th.—She passed a motion not unlike that of typhoid of fever; it was bright yellow in colour, with streaks of blood, alkaline in reaction, but without much odour; it was mostly liquid, with a few scybala. The abdomen was much distended, tender at the lower part. There were no spots. The motions soon became more normal, and the bowels were then constipated.

November 9th.—Optic neuritis not increased, worse on right than left side; right retinal veins very tortuous.

During the latter part of November she again had much abdominal pain, and anæmia continued to be a marked feature.

In January she had cough, and some bronchitic râles were heard in the chest. She became weaker, paler, more apathetic, and even drowsy, requiring to be roused to take food, but still complaining of some abdominal pain.

On the 14th it was noted that the spleen had much enlarged since the beginning of the month; it reached down to the level of the umbilicus, and the liver projected three quarters of an inch below the right ribs. Her cough continued troublesome, and she was unable to sleep. She got progressively weaker, emaciated rapidly, the temperature fell below normal, and she died on January 21st, six months and two days after coming into the hospital.

The record of the temperature between August 8th and September 30th has been mislaid; but from this date to a few days before her death it presented daily oscillations of considerable regularity; that is to say, the lowest morning temperature was, as a rule, at or below 99°, and the highest evening temperature was generally above 101°. The difference between the two was at least 3°. During the month of December there was a tendency for the fever to be higher. The last really high temperature was on January 15th; it fell during the 16th and 17th to normal, and the last four days of life was subnormal. The pulse during these four months was mostly from 120 to 140; the respirations from 28 to 34.

Autopsy by Dr. Pitt.—The *brain* contained a slight excess of cerebro-spinal fluid beneath the membranes; those covering the second and third left frontal convolutions, and the central convolutions on both sides, were ochreous in colour from blood which had been long extravasated. There was no other change obvious, except that at the tip of the left temporo-sphenoidal convolution there was a depressed scar, irregular, about half an inch along, and radiating in several directions. There was a slight thinning of the cortex at this point. There were about ten minute fibroid nodules beneath the pia mater from ½ to 1 mm. in diameter; they were discrete and far from one another. There was no inflammation near them, and their nature is obscure. The vessels of the brain appeared normal. Weight 38 oz.

Pleuræ.—Serum, 4 oz. in the right, and one in the left pleuræ.

Lungs.—Typical heart-lungs; a patch of early broncho-pneumonia at the middle and posterior part of the left lung. The central portions of the lung were airless, and sank more readily in water than did the peripheral portions. The bronchial glands were swollen.

Heart.—Weight 8½ oz. (normal weight for this age 4½ oz.). Right side slightly dilated; apex still formed by the left ventricle. Left ventricle markedly dilated and hypertrophied. Fungating endocarditis of the mitral valve; some of the chordæ tendineæ ruptured and rolled up into knots with lymph. The margin of the valve towards the auricle was covered with numerous projecting masses of organising clots. These were decolourised and tough and of some date. There was an area about one and a half inches across on the left wall of the auricle, extending to the valve, which was covered with numerous small projecting nodules of organising vegetations. These had been formed partly by the flapping of the ruptured cords, and partly by the regurgitant stream.

The rest of the auricular endocardium was opaque and thickened. Microscopically, these vegetations were on their surface covered with innumerable micrococci aggregated in dense groups. They appeared to be staphylococci, rather than streptococci, for nowhere could any strings be found.

The other valves were normal; and to the eye the cardiac muscle appeared normal.

Pericardium.—Normal.

The peritoneal cavity contained 18 oz. of fluid. The *liver* weighed 33 oz., and was healthy, not nutmeggy in appearance, with several superficial patches of fatty infiltration, sharply defined, about two or three inches across. These were confined to the area in front of the right kidney, and did not appear to be embolic.

The *spleen* was very large, weighing 15 oz. There was a chronic capsulitis, with several large infarcts of various date. Some of these were nearly white in colour, caseous and indurated round the margin. Others were recent, dull red in colour, and not so dense as the former.

Kidneys.—6 oz.; there were several infarcts, most of them old scars, leaving depressed areas; one still with a line of caseous material at the surface. In addition there were some larger and more recent infarcts, one of which had not begun to absorb; the centre was necrotic and the margin hyperæmic. Some of the glomeruli were marked out with petechiæ, and there was mottling of the tissues, so that there certainly was some, but not much, acute nephritic change.

CASE 4. *Mitral endocarditis; former rheumatism; pyrexia; albuminuria; anæmia; optic neuritis; convulsions; lardaceous spleen, with infarct; lardaceous inflamed kidneys.*—Margaret M—, æt. 38, was admitted under my care on October 10th, 1887.

She had rheumatic fever when sixteen years of age, and also about two years ago, when she was laid up for three months. About seven years ago the nose used to bleed occasionally. Last January she caught cold and had shivering now and then, and she has continued weakly and feverish since. Two months ago she noticed red spots on her legs; they disappeared and came again, and she has not been free from them since. Lately some of them have felt painful when touched. About a fortnight ago she became very short of breath whenever she exerted herself in any way. Sometimes her heart used to palpitate a good deal, and her head ached. Her appetite has been good. The gums have never been affected.

On admission.—She is anæmic and weak, but free from pain while she is resting. On both legs are numerous hæmorrhagic spots, varying in colour and size, some being petechiæ, others amounting to vibices. The legs are not now swollen, but they have been. The lips and gums are pale, the tongue dry; the bowels are regular. The lungs appear nearly normal; resp. 20; no cough. The heart's apex beat is felt in the fifth space, one inch directly below the nipple. The cardiac dulness is normal. There is a loud systolic bruit at the apex, heard towards the axilla, but only faintly towards the base; and here the murmur is louder over the pulmonary artery than over the aorta. No murmur is heard behind. Hepatic dulness reaches two inches below the costal margin in the nipple line, beginning above at the sixth interspace. The liver is painful on percussion. The spleen is enlarged, extending in front below the edge of the ribs, and it is tender on percussion.

Urine, sp. gr. 1022, contains no blood, nor albumen, nor sugar. The temperature was 100·4° on the evening of admission.

She has not menstruated since January.

The patient continued weak, without other notable complaint, and she had an oscillating temperature, which reached 101° or 102° in the evening, and fell to 99° or 98° in the morning. On October 25th there was thin line of albumen on adding nitric acid to the urine. On the 28th I found double optic neuritis.

On this day also she had a slight fit, in which the left arm was convulsed, and the right slightly so; it continued three or four minutes; she remembered nothing of it afterwards. On October 3rd petechiæ appeared on the chest and a few behind the left shoulder. The hæmocytometer showed that white corpuscles were in proportion to the red as 7 or 8 to 100. The spleen could be felt 2½ inches vertically from the edge of the ribs in the nipple line. It was very tender.

On November 1st the purpuric spots were fading, and were almost gone on the 3rd. On November 4th she had another fit, in which she was unconscious and screamed. The face and limbs were convulsed, especially the face, and the eyes were turned to the right. The fit lasted some minutes. On November 5th she was very dull, and could with difficulty be persuaded to take food. The urine was smoky, neutral in reaction, contained blood, albumen, a large number of blood corpuscles, and granular casts. She died the following day at 6 a.m.

There was no change in the murmur at any time; and no glands were observed to be enlarged. The report contains no record of temperature beyond October 18th.

The *autopsy* was made by Dr. Perry:

The *heart* weighed 13 oz. There was much fat on the right ventricle and and at the apex of the left, and the right ventricle showed much fatty infiltration. There were no petechiæ under the pericardium.

Mitral valve.—The edges were thickened and puckered; a vegetation, the size of a pea, on the anterior flap, and one of less size on the opposite point of the valve; and above this, on the auricular endocardium, a roughened surface, as from friction of the vegetation against it. The circumference of the orifice was nearly four inches. Aortic valves healthy. There was no ulceration on the right side. A piece of the mitral valve, cut and stained with methyl violet, showed no more than a roughened surface, and beneath it a good deal of leucocytal infiltration.

The *liver* was normal. The *spleen*, 17 oz. was soft and slightly lardaceous; it contained an old infarct of considerable size.

The *kidneys,* 19½ oz. had a white cortex with well-marked stellate veins; the pyramids dark and congested. Methyl violet showed slight, but almost universal, lardaceous change in the vessels. The degeneration of epithelium was shown to be considerable. Thickening of Bowman's capsule occasionally present; some glomeruli much dilated; vessels not thickened, and only lardaceous just at their entry into the tufts. Tubes compressed and destroyed by enormous interstitial growth. Some tubes dilated with flattened epithelium, some blocked with proliferating epithelium, some containing large homogenous colloid casts with indented edges. The medulla showed no lardaceous change in the vessels, but much increase of connective tissue, and small cell infiltration, with destruction of tubes.

The *brain* weighed 49 oz. and was healthy.

CASE 5. *Mitral and aortic endocarditis; pyrexia; "typhoid" motions; orchitis; inflamed joints; infarcts in spleen and kidneys.*—J. T—, æt. 32, was admitted under my care into Stephen Ward, Guy's Hospital, on September 8th, 1884. His work consisted in cooking sheep's feet in large quantities in coppers, so that he was exposed to great heat, and frequently, on returning from his work, had shivering fits. His father is well, æt. 73. His mother suffered from ununited fracture. Four brothers and sister are well.

He had rheumatic fever five years ago, and has been told by a doctor that he has heart disease. He often complains of his limbs and is subject to colds. He has been married six years, and has three children, of whom two are living, but are not strong.

He was at work until Monday, September 1st, when he came home early, complaining of pains in his back between the shoulders; and he had several shivering fits. He took to his bed on Tuesday, and suffered from shortness of breath and great thirst. He has scarcely taken any solid food since the commencement of his illness. On Thursday, the 4th, Dr. Marshall, the parish doctor, saw him, and prescribed a saline purge, together with calomel and jalap. On Saturday he advised his coming to the hospital.

On admission.—His temperature was 103·2°; pulse 100;

respirations 40; alæ nasi acting; skin dry; tongue dry, red at tip and edges, covered in the middle with brown fur.

Moist sounds were noted at both bases in front, and the heart's action was very rapid. The bowels were opened twice on the day of admission, but the motions were not loose.

On enquiring into his surroundings, it appeared that there was no epidemic in the neighbourhood. He was ordered five pints of milk; brandy 4 ounces.

At 5.30 p.m. he had an anxious expression, was sensible, but flurried and nervous. The muscles all over the body were twitching. Skin dry and hot. The temperature was 103·8° in the evening, and fell to 102·8° at midnight.

September 9th.—Passed no urine till 1 p.m. to day, and then passed it in bed. Motions to-day were of fluid consistency, and of bright yellow colour. The abdomen is much distended; not tender; no spots. The left side of the scrotum is red, and the testicle is swollen and very tender. Temperature in the early morning was 101.8°, it rose to 103·2° in the afternoon, and was 102·8° at midnight. Pulse from 124 to 132. Resp. 36 to 40.

September 10th.—Passed three motions in the night, regarded as "typically typhoid." This morning he lies on his back, with very anxious expression; hurried breathing; tongue covered thickly with moist white fur. Answers when spoken to, and does what he is directed, but is constantly muttering "Oh, what shall I do?" The temperature was between 102° and 103° all day; his bowels were open about midnight.

September 11th.—Delirious this morning. Screams on being washed; passes everything under him. The distension of the abdomen much less; the spleen cannot be felt; there are no rose-spots, but a few petechiæ over the chest. The heart's action is very rapid, markedly irregular, but no bruit is audible on auscultation. The impulse is felt three quarters of an inch outside the nipple, and one inch below. Orchitis is less; he perspires profusely. The tongue is covered with moist white fur; sordes on lips. Reflexes normal.

At 3.30 p.m. he was nearly unconscious, though there was obvious tenderness of the right arm, mostly about the wrist and elbow-joint; but he is generally hyperæsthetic. Petechiæ are noticeable over the chest, forearm, and back of right hand. There is some bronchitis. The temperature was 102·8° at 6 a.m., fell to 102° at 3 p.m., and rose in the evening to 103·4°. He was ordered 15 minims of tincture of digitalis in ammonia mixture every three hours.

September 12th.—He has had a very bad night. Has been sponged four times since 9 p.m., but without any great reduction of temperature. At 9 a.m. it had risen to 105·8°. The bowels were open three times in the night. He is now quite unconscious, lying with half-closed eyes. The petechiæ are numerous over the right shoulder and chest; there are no typhoid spots. The left wrist-joint and elbow-joint are very tender. The orchitis is about the same. The abdomen is distended. There is a symmetrical eruption over the backs of both hands and forearms, and on the dorsum of each foot; it is red, diffused, erythematous; not raised; disappears on pressure. The temperature continued to be high all day. It was 105·6° at 9 a.m., 104·2° at 12, 104·4° at 6 p.m., and 105° at 9 p.m. On each of these occasions it was reduced two or more degrees by sponging. He died at 11.10 p.m.

Autopsy.—Pronounced rigor mortis and hypostasis. The right *pleura* was adherent to the parietes at the base; the left was also adherent to the chest wall and thickened. Both *lungs* were congested, especially at the base, and œdematous, and pus was present in the smaller tubes. The *pericardium* was completely adherent to the heart everywhere; and coarse filaments stretched between it and the adjacent pleura. The adhesions separated with some difficulty. In the left auricle there was no friction lines nor vegetations. The mitral valve was thickened with many vegetations, for the most part small, and situate at the free edges. The chordæ were intact. The endocardium of the left ventricle just below the valves was thickened, and showed a granulating patch about the size of a threepenny-piece. On the free edges of the aortic valves were some small granulations. The right side was quite normal.

The large vessels were healthy. The small intestines were quite healthy, without trace of any ulceration and swelling of Peyer's patches and solitary glands. Contents bright yellow. *Liver* normal. *Spleen* enlarged, soft, with several partially decolorised infarcts, not softening. *Supra-renal capsules* normal. *Kidneys* swollen, pale, mottled on the surface, with many small decolorised (yellow) infarcts and one or two larger ones; none softening. *Bladder* healthy. *Left testicle:* purulent lymph in the digital fossa. The epididymis was not swollen or hard; there were no signs of inflammation in the vesiculæ seminales, or any trace of inflammation of urethra. There was no pus in the left shoulder-joint, nor in the left elbow; but on making an incision in the neighbourhood of the joint some oozed out, but from what point could not be ascertained.

CASE 6. *Chorea; mitral endocarditis; pyrexia; petechiæ; retinal hæmorrhages; "typhoid" stools. No autopsy.*—John F. G—, æt. 6, was admitted into the Evelina Hospital under my care on November 27th, 1884. His father was alive, but his mother had died of acute rheumatism; and there was a strong history of rheumatism in the mother's family. On the father's side an uncle had had rheumatic fever.

This child had measles and whooping-cough when eighteen months old. Five months ago he fractured his right clavicle. His present complaint dates from two weeks ago, when twitching movements were first noticed, and they have gradually got worse. He has also complained of pain in the right side. He is not known to have had any fright.

On admission.—He is a child with fair complexion. There are slight choreic movements, most noticeable in the hands and arms, and least in the lower extremities. He can feed himself. The tongue is fairly clean. Temperature was normal on admission. There is no pain in any of the joints. The cardiac impulse is in the fourth space just under the nipple; no increase of cardiac dulness; a very soft bruit at the beginning of the first sound, heard only at the apex; there is no thrill.

The lungs, spleen, and liver appear to be normal.

November 28th.—The temperature rose to 103·2° at

3 p.m. The tongue is furred, white. There is no diarrhœa; no rash; no pain; no sickness.

At midnight the temperature fell to 97·4°, rose again in the course of the next day to 103·2°, and fell again at 9 p.m. to 99°. He complained of headache, had a flushed face, slight cough; and the bowels were confined, so that castor oil, and subsequently an enema, was given. The bowels were moved three times.

30th.—Complaint of abdominal pain. Slight cough. No sweating. Temp. 2 a.m., 103°; 6 a.m., 99·6°; 12 noon, 101·6°; 3 p.m., 99·4°; 9 p.m., 103·4°; midnight, 97·4°. Ordered one grain of quinine every four hours.

December 1st.—The abdomen is moderately distended; there is no sore throat, no diarrhœa. Some gurgling in the right iliac region. Face flushed, tongue moist and white.

4th.—No change in the general symptoms. The temperature continues to be of the intermitting type, varying from 98° to 104°. There is no diarrhœa. The nurse thinks she recognises a typhoid smell.

Between the 6th and the 11th the temperature had a less decidedly intermittent character, and the oscillations, of which there were three or four in a day, were rarely more than 2°, ranging between 100° and 103°. On the 11th there was a difference of 3·8° between two observations, and on the 12th a sudden rise of 4°, and these wider excursions continued till the 18th, three days before death.

15th.—The report says:—"He is now having typical typhoid stools, and complains of much pain in the abdomen. He has a few petechial spots on the inner side of the left thigh, on the scrotum, and on the lumbar region behind. The temperature is highest in the afternoon and evening. The respirations are twenty-four to the minute; the pulmonary sounds fairly normal, except at the bases, where there is a little crackling. The heart sounds have altered since admission; the systolic bruit is much louder, and is no longer localised, but can be heard in the axilla. The second sound is accentuated. Pulse 140. He moans with pain, which he refers to the epigastrium. No pain in the joints. He has been taking daily—milk, two pints; broth, one pint; brandy, three ounces. Hitherto he has taken this well, but he has vomited to-day, and the broth is omitted. Tongue moist, slightly furred. Ordered yesterday Tr. Opii ♏v ex Aquâ omni nocte. On December 19th he was ordered Tr. Ferri Perchloridi ♏vii, Potass. Citratis gr. v, Syrupi Limon. ʒss, Aquâ ad ʒij, t. d. s.

20th.—He is apparently blind with both eyes, though the pupils react slightly to the stimulus of light. There are large hæmorrhages in both retinæ; no neuritis. He has internal strabismus on the right side, but this is due possibly to his well-marked hypermetropia. The trunk is covered with small petechial spots. The abdomen is less distended; he continues to have motions like those of typhoid. The mouth and tongue continue comparatively clean and free from sordes. The pulse is feebler, 144. He now takes three ounces of brandy. He still has a good deal of pain when the effects of the opium have passed off, and he is not always quite conscious.

21st.—Died at 8.40 a.m. His temperature had fallen to 97° at 3 a.m., but rose again to 100° before death.

No inspection was allowed.

Case 7. *Ulcerative endocarditis; mitral regurgitation; enlarged liver and spleen; hæmaturia; epistaxis; retinal hæmorrhages; severe anæmia.* (From notes by Mr. F. Wellford).—Henry C. K—, æt. 34, was admitted under my care on March 6th, 1889, for pain in the cardiac region, over the right side of the chest and back; for cough, occasional spitting of blood, pains in the legs and swelling about the ankles.

Life history—The patient was born in Limehouse, and lived in London as a working man till he was twenty-eight. He went to sea as a coal-trimmer, and spent six years on the Baltic, Mediterranean, and American waters. He left the sea last July, and from then to December 21st worked in a jam factory. He has been out of work since December 21st, and has not lived so well since. He married in 1882, and has two children.

Family history.—Father alive, fifty-nine; rheumatic, paralysed. Mother alive, sixty; supposed to have been consumptive when young. One of his mother's brothers died from consumption. Three sisters, all healthy, except one, who is not strong; patient does not know if there is anything definitely the matter with her. Two brothers, both healthy.

Personal medical history.—When about eight years old patient was treated at the London Hospital for St. Vitus's dance and heart disease. He gradually got better, but has always been troubled by palpitations and pain about the heart, especially when lifting weights and exerting himself much. He has always been subject to colds and sore throats. He was three months in King's College Hospital when about eighteen for heart disease. When twenty-one was in Guy's, under Mr. Durham (Cornelius Ward), with a broken jaw. From the report then made it seems that the wound gave great trouble by constantly bleeding during the first eight days after his admission. When twenty-eight he had a fall from a tram-car, and was treated at St. Thomas's Hospital for concussion of the brain. Four years ago he had an attack of dysentery at Galaty, and was ill all the voyage home to Antwerp, where he was landed and sent to a hospital. Two years ago he fell forty feet off the end of a jetty, and was in consequence in the London Hospital for three weeks; he did not sustain any permanent injury. The last year that he spent at sea he began to feel slack and out-of-sorts, and disinclined to do his work, which, he says, was very unusual with him. In October last he found his work too much for him. He first noticed attacks of pain in his back, then a constant dull gnawing pain over the heart; a little later he vomited up his food, and about the same time began to have a cough, and now and then to spit up a little blood. Since his discharge on December 21st he has got steadily worse. The pains are more frequent, and the cough more constant; the vomiting after food has ceased, but his cough often makes him retch. His ankles and feet swell at times, and he is much troubled with night sweats. He first noticed these last two troubles at the end of December. One day last week, during a fit of coughing, he felt a sharp sudden pain through his heart. He has shivering fits occasionally.

General appearance.—The patient is a fairly well-made man. His arms give one the impression that he has been tolerably muscular, but he is now thin and extremely pale. Eyes grey; the conjunctivæ almost colourless. Skin smooth, hot, dry, very pale. There is an enlarged lymphatic gland, about as big as an almond, near the middle of the posterior border of the sterno-mastoid muscle, on the right side of his neck. No œdema of the ankles; on the inner side of the left knee are some pigmented marks.

The cardiac impulse can be felt in the fifth interspace about 1¾ inches below the left nipple and in the nipple line. Pulsation can be seen in the fifth interspace, a little below the left nipple, and also at the root of the neck. The cardiac dulness is normal. On listening over the apex a loud blowing murmur can be heard replacing the first sound; it is best heard ¾ inch internal to the nipple line, but can be traced round towards the back for three inches. A similar sound is heard at the angle of the scapula, but much fainter. A murmur is heard on listening over the vessels at the root of the neck. Pulse 72.

The patient has a dry cough, which comes on about bedtime; expectoration frothy; no mucus; no pus; no blood. Chest fairly deep and broad. Breathing chiefly thoracic. Movements the same on both sides; vocal fremitus normal. The percussion note just below the clavicle is very slightly less resonant on the right side than on the left; the breathing is perhaps rather harsher, and there are a few obscure râles, but there is no bronchial breathing.

Lips of usual colour. Teeth very irregular, broken, and carious; gums rather pale; tongue normal. The patient says that he finds mastication difficult owing to his bad teeth; he can swallow without trouble, and does not now vomit up his food. His bowels are regular, and he does not pass blood with his motions.

He complains of pain over the right hypochrondriac region, and of tenderness on pressure in this region. On percussion there is a dull note over the region of the liver. This note persists over an area bounded above by the sixth rib, and below by a transverse line drawn through a point ½ inch above the umbilicus. The edge cannot be distinctly felt, but there is a sense of increased resistance as the hand passes from the resonant to the dull area.

The contraction of the abdominal muscles renders palpation of the spleen difficult; its dulness appears to be increased.

The patient is quiet and intelligent, but his memory does not seem good. The pupils react to light and accommodation. The optic discs are normal. The patient is rather deaf on both sides.

Urine.—Rather high coloured; acid reaction; sp. gr. 1018, no albumen, sugar, or blood.

Temperature 99·2°.

March 10th.—He was ordered digitalis, iron, and quinine, and pills of zinc oxide and belladonna at night.

13th.—The heart sounds have not altered in character. The pain and tenderness over the hepatic region are about the same; there is perhaps a diminution of pain in the cardiac region, but the patient complains of a gnawing pain on the left side, in the region covered in by 10th, 11th, 12th ribs. The night sweats have not been so profuse the last three nights; last night he hardly sweated at all. No alteration in the cough. The four-hour temperature chart for the week shows a regular remittent pyrexia, varying between 98° and 103°; it is highest about 10 p.m., and lowest at 10 a.m. No blood or albumen could be detected in the urine this morning.

Examination of the blood on the 11th showed a considerable deficiency of red blood-corpuscles, but no increase of leucocytes.

16th.—The patient complains very much of a sharp stabbing pain, increased by forcible expiration, in the right side of the chest, over an area whose upper margin is an inch below the nipple.

On the 18th he was ordered four grains of antifebrin daily at 1.30 p.m., but on the 21st there had been no deviation from the usual course of the pyrexia.

On the evening of March 22nd the motions were loose and yellow, though not copious, and they were again loose the next morning, the night of the 23rd, and the morning of the 24th. During this time he was feeling very unwell.

26th.—In much the same condition. There were slight traces of albumen in the urine this morning; no blood or casts. The urine is examined every day; this is the first time that any albumen was detected. Sp. gr. 1020, colour rather high, reaction acid.

29th.—The patient complains of a gnawing pain in the right loin this morning; he says it came on suddenly when he turned from his back to his left side. He complains of great tenderness on pressure, both when the pressure is applied behind, just below the twelfth rib, and at a corresponding place in front; he complains of a similar, but not so severe, pain in his left loin. The heart sounds are unchanged; pulse is 80, and can be distinctly felt at the wrist, in the temporal, facial, internal tibial, and dorsalis pedis arteries. The last can be seen pulsating in both feet. The characters of the temperature chart are unchanged, except that for the last week the maximum point has been about 102° instead of 103°. The minimum is still about 99°. The urine contains traces of albumen; no blood or casts; sp. gr. 1020, colour high, acid.

April 1st.—Much the same. The urine shows traces of blood and rather more albumen this morning. Cloudy from mucus and phosphates. Sp. gr. 1010, acid.

8th.—The patient's condition has not changed. Since April 1st albumen and blood have always been present in urine. The patient got up and lay on the couch a little yesterday afternoon; he fancies he slept better in consequence. He is certainly more cheerful this morning.

10th.—On asking the patient to read with the left eye alone, he does so with difficulty, complaining of a mist. He can read easily with his right eye, in which he says there is no mist; he says that a lamp on the table looks like a blood-red mist to his left eye. The ophthalmoscope shows a hæmorrhage in the yellow spot of the left eye. His blood was examined to-day, showing red blood-corpuscles 40 per cent. of normal.

Antifebrin discontinued; hæmoglobin 32 per cent. of normal. Sodium sulpho-carbolate ordered in ten-grain doses.

15th.—The patient passed a very bad night; very excited, delirious, and shouting. Urine, sp. gr. 1012, acid, with blood, albumen, and granular casts.

16th.—There are hæmorrhages in both eyes. In the right eye there is a red flame-shaped hæmorrhage, starting from the upper edge of the optic disc, rather on the nasal side. In the left eye the hæmorrhage is some little distance outside the disc, almost directly above the yellow spot; it is almost black.

17th.—The patient insisted on going out to-day. He is certainly no better than when he came in. The heart sounds are the same as on the day of his admission. Pulse this morning 108. Pulses were felt on the 15th in the temporals, facials, at both wrists, in the dorsalis pedis; equally distinct on both sides.

The liver and spleen give the same signs of enlargement as on admission. The lungs show no signs of disease, the chest moving equally on both sides, and the air entering well. Resonance good. The patient's tongue is clean and his appetite good; his bowels are regular. His mind is clear, but he speaks slowly, and every now and then has some difficulty in remembering a word, especially if it is a name. He is rather irritable and melancholy. Temp. 97·4° at 6 p.m., 101·2° 10 p.m., 16th.

Urine contains albumen, blood, and granular casts.

The patient's appetite throughout has been good. He is thinner than when he came in, and walks more feebly. He has had a slight cough throughout, with a little frothy serous sputum. Occasionally this has been tinged with blood; but this has always been in association with slight epistaxis, to which it may perhaps be referred.

Subsequent history.—This patient died in the London Hospital some time in the third week of July, 1889. The following notes were kindly supplied by the Medical Registrar of the London Hospital:

Post-mortem.—*Lungs.*—Œdematous, congested, with some bronchitis on squeezing. Some emphysema, especially at the left apex, where there was some cicatrisation.

Heart.—All valves healthy except the mitral, the posterior curtain of which was involved, together with the posterior wall of the left auricle, in what was evidently recent acute endocarditis; it was covered with recent vegetations.

Kidneys.—Congested (?); some slight acute nephritis.

Liver.—Congested. No other pathological naked-eye change in any other organ.

CASE 8. — *Rheumatic fever and heart disease; aortic and mitral endocarditis; pyrexia; hemiplegia; infarcts in spleen and kidney.*—Charles R—, æt. 27, was admitted under my care on May 14th, 1887, for paralysis of the left side.

He had rheumatic fever when 12 years old; and he was told by his doctor that his heart was affected.

Two months before last Christmas he fell downstairs with a child in his arms and hurt his back. Four weeks after that he had pain in his left side. He was better at Christmas, but got a cough after that, and occasionally perspired freely at night. Shortly after this he had an attack of "gout" in the left foot, lasting one day. Nine weeks ago he had again severe pain in the left side—"bronchitis and pleurisy," according to his doctor.

On April 25th, at 5 a.m., having eaten rather freely the day before, and suffered from pain and sick-feeling, he was found to have paralysis of his left arm, leg, and face. At 11 a.m. he vomited. He passed everything under him for two or three days, and bedsores soon formed on his back and left thigh.

On admission he is emaciated, anæmic, with black hair and dark eyes. He generally lies with the head directed to the right, and is drowsy or even snoring. If roused, he can answer questions slowly, but distinctly. He talks with some difficulty, and his voice is husky.

He cannot move the left arm or leg; the left angle of the mouth is dropped, the tongue protruded to the left; the left eye does not close so tightly as the right; and the left forehead does not wrinkle on elevation of the eyebrows. He can feel, and localise touches on the left side. There is no conjugate deviation of the eyes. Knee-jerks present and equal. Plantar reflexes present and equal. He has three bed-sores, one on the middle of the lower part of the back, one over the left trochanter, and one lower down on that side.

Pulse 120, regular, of rather slapping character. The heart's beat is forcible in the fifth space, one inch below and one inch outside the nipple. A systolic murmur is heard at the apex, preceded by an indistinctly double second sound; and there is a slight systolic murmur in the right second space.

Respirations 32. Impaired resonance at both bases behind from the ninth ribs downwards, and air enters badly. He has a troublesome cough. Hepatic dulness extends a finger's breadth below costal margin. Splenic dulness normal. Urine of sp. gr. 1020, slightly acid, clear, dark amber; no albumen, sugar, or blood. Optic discs normal.

On May 20th a musical, humming diastolic bruit was heard over the left third intercostal space.

On May 23rd the click of the pulmonary valves could be felt in the first left space, and it was noted that the heart's impulse could be felt over a space limited below by the upper margin of the fifth rib, and internally by the left margin of the sternum; there was pulsation also in the fourth space external to the nipple.

The humming diastolic murmur could be heard, and at the apex a slight presystolic bruit. The temperature hitherto had been mostly subnormal; on the evening of the 23rd it rose to 102·6°, and then fell to 101·6°.

On May 25th the bedsores were healing; the urine was free from albumen. On the following day a few râles could be heard in the front of the chest on the left side. On the 25th some segments of tapeworm were passed, and a dose of male fern administered on the 27th brought away six yards of tænia. On the 30th he was breathing somewhat fast, and about 6 p.m. the nurse found that he was paralysed on his right side.

He died on the evening of the 31st.

The temperatures recorded during the last week of his illness were as follows:

May	24th.—M.	96·6°	E.	100°.
,,	25th.—M.	97°	E.	100·6°.
,,	26th.—M.	97·8°	E.	101°.
,,	27th.—M.	97·8°	E.	101°.
,,	28th.—M.	97°	E.	99·8°.
,,	29th.—M.	97°	E.	99·6°.
,,	30th.—M.	96°	E.	102·6°.
,,	31st.—M.	97°	E.	99·4°.

and it sank to 96° before death.

Autopsy by Dr. Pitt.—Pericardium adherent. *Heart* hypertrophied, weighed 17 oz.; the cavities somewhat dilated. Papillary muscles extremely fibrous.

Mitral curtains thickened; orifice only admitted two fingers; the edge, especially on the auricular surface, was covered with granulations the size of millet seeds, white in colour, rather soft, and evidently for the most part of recent date. The aortic valves were slightly thick. The anterior valve had a mass of yellowish-white vegetations along the lunula on the right half, one sixth of an inch thick. There was a small nodule of vegetations on the right posterior valve at the corpus Arantii, and on the left posterior segment was also a mass of vegetation; the vegetations were all more or less recent; none tough and calcareous, and none were ulcerative, or even deeply blood-stained, so that the condition could not be spoken of as one of ulcerative endocarditis. There were no ulcerating friction patches on the walls of the heart.

Lungs.—At the right base a depressed portion like carnified lung; adjacent to this were areas with overdistended alveoli. Slight œdema; no inflammatory changes except a little muco-pus in the larger tubes.

Liver slightly congested. *Spleen* 17 oz. Large, firm, with several large creamy-white infarcts, with no evidence of softening or suppuration. *Kidneys* 11 oz.; one small, pale infarct, with extremely congested pyramid in corresponding part, otherwise healthy.

Brain.—A decolorised clot in each middle cerebral artery, that on the left side recent, that on the left old. White softening was found on the right side in the pediculo-frontal, frontal, parietal, and pediculo-parietal sections, occupying the external capsules in the second, and the internal and external capsule in the third. There was also less obvious white softening on the left side, in the frontal (external capsule), parietal (internal capsule), and pediculo-parietal sections.

There was no hæmorrhages, and no secondary degenerative changes visible to the naked eye.

CASE 9. *Endocarditis involving mitral valve; pyrexia; rigors; infarcts in spleen.* (From notes by Dr. T. F. Ricketts.)

—Edwin Henry J—, æt. 39, was admitted under my care on June 30th, 1888.

He is a printing-machine manager. He has not had rheumatic fever, but is subject to "rheumatics." Two years ago he was laid up with an ulcer of the right leg, probably in connection with a vein. He is said to have had sciatica. His wife died in February of diphtheria.

He was taken ill on June 16th, though he had not been feeling quite right for some days. When he returned from work on that day he felt very ill, and went to bed. His doctor told him he had rheumatics in his foot; and he is said to have had pain in the right foot two days before this. He continued to be ill, with pain all over him; cough and expectoration, some blood being mixed with the phlegm. He became strange in his manner on the day of admission.

On admission.—The patient is wasted, with sunken cheeks and temples; he is bathed in a profuse perspiration. He lies in a half stupid condition and dozes a good deal; he recognises his friends, but rarely speaks unless spoken to. He appears to understand what is said to him, but he answers after a long interval, if at all, and his speech is slow and indistinct. His remarks are often irrelevant, and his memory is at fault.

The limbs are wasted but well formed; there is no pain or swelling of the joints. Two scars are seen on the right leg, none on the penis.

The chest presents slightly diminished resonance on the right clavicle and in the supra-spinous fossa; hyper-resonance in front extends over the precordial area. There are a few small râles at both extreme bases behind, and in the left supra-spinous fossa. No coughing, no expectoration, and no dyspnœa.

The heart's dulness is absent; the impulse neither seen nor felt. A systolic murmur is heard at the apex, in the axilla, and behind; at the base the sounds are muffled.

Tongue moist, furred. Breath rather foul. Abdominal walls rigid; liver dulness reaches two inches below the ribs. There is no paralysis and no squinting. He is deaf in the right ear, but there is no discharge.

He had a rigor on the night of admission, another on the following morning, July 1st, and he vomited on the morning of July 2nd.

July 2nd.—Pulse 120; resp. 35.

3rd.—Another rigor this morning, the temperature rising to 104°; profuse sweating afterwards. While he dozes the right eyelid does not close so well as the left, and the left angle of the mouth appears slightly depressed.

He has had several liquid motions since admission; they contain neither pus nor blood.

5th.—Patient has become semi-comatose; does not speak, and if spoken to, at most mutters indistinctly. He had a rigor at 11 a.m. Later, he was lying on his left side, with mouth half open, and his eyes open, with vacant look. His pulse now reached 160, and respirations 48, with an indistinct Cheyne-Stokes' rhythm. He became more sensible towards evening. Liquid motions continue, and are passed under him. No increase of splenic dulness, nor is the spleen felt.

I examined the optic discs and found them normal. The right ear is blocked by cerumen.

6th.—Diarrhœa continues, and the motions are streaked with blood; but he has internal piles.

7th.—Diarrhœa continues; he is more rational.

8th.—Still improves mentally. No vomiting for two or three days. The urine is acid, sp. gr. 1020, slight deposit of mucus, no albumen.

9th.—Not so well; takes food less readily; scarcely speaks; diarrhœa persists.

11th.—The systolic bruit appears to have a whistling character added to it, especially noticeable in the seventh space. Papules, possibly hæmorrhagic in character, noticed over the left patella. No sign of paralysis; knee-jerks brisk; ankle-clonus.

13th.—A slight rigor at 11 a.m. Increased stupor. The murmur presents a somewhat musical character.

14th.—Semi-comatose; excretions passed under him; extremities cold. In the evening tubular breathing was heard at the left base. More spots, some of which were distinctly hæmorrhagic, came out near the right patella and on the right thigh.

15th.—Death at 5.30 a.m.

The temperature was irregular, and interrupted by the rigors. The four-hour chart showed that the mean was considerably above the normal, but there were not such regular morning falls and evening rises as are often seen. In seven of the twelve days of which there is a full record, the temperature reached 101° or more, and on the other days the highest point was 100°. On seven days also the lowest temperature was about 97°.

Autopsy.—Petechiæ on both pleuræ, mostly behind and at the lower parts of the lungs. *Lungs.*—Emphysematous, with congested and œdematous bases. *Larynx.*—Ecchymoses beneath the mucous membrane.

Pericardium.—A few petechiæ beneath the visceral layer. *Heart.*—11 oz., muscular fibre pale, no infarction. The mitral valve showed old thickening along the free border, but had probably been competent; the left auricle contained a large clump of vegetations, the size of half-a-crown, and a quarter of an inch in thickness, situated in part upon the auricular surface of the valve, the rest being adherent to the endocardium. Aortic valves unaffected. Right side of the heart normal.

Œsophagus.—Petechiæ beneath the epithelium. *Large intestine.*—Matting and thickening of the parts about the cæcum, the vermiform appendix being adherent to the intestine. On opening the cæcum there was puckering of the mucous membrane, and about four inches below the ileocæcal valve were two irregularly rounded ulcers, each about the size of half-a-crown piece, with raised thickened edges; no tubercle was seen. The mucous membrane at the puckered spot showed very large and engorged veins, and there were one or two projections of mucous membrane, like polypi, each containing a black dilated vessel, the largest hanging half an inch into the intestine. The rest was normal.

Liver.—67 oz., seemed healthy. *Spleen.*—9 oz., large and soft, contained two large decolorised infarcts. *Kidneys.*—9½ oz., of ordinary size, and appeared healthy. No petechiæ, no abscess, no infarcts; one small fibroma.

CASE 10 (Mr. Golding-Bird, 1885).—George K—, æt. 62.

Admitted for aneurysm of profunda femoris artery. Systolic murmurs at apex and base. Ligature of external iliac. Pyrexia, pneumonia. Death five days after the operation.

Autopsy.—Endocarditis with vegetations on aortic valves causing constriction. Pleurisy and pneumonia. Fatty liver. Old infarcts in spleen. Kidneys fatty, with wasted cortex, cysts and two scars of former infarcts.

Case 11 (Dr. Wilks, 1885).—Margaret D—, æt. 8. Has had measles, whooping-cough, scarlet fever, and bronchitis; in bed two years. Enlarged heart, with mitral regurgitant bruit, increased præcordial dulness, and pericarditis. Consolidation of base of left lung. Temperature between 98° and 102° or 103° until a few days before death.

Autopsy.—Fluid in pericardium, and old cord-like adhesions, thickening of mitral cusps with long granulations, adherent fibrin, and chordæ separated. A small vegetation on the tricuspid valve. Indurated lungs. Two recent infarcts in the spleen, and two scars of infarcts in the kidneys.

Case 12 (Dr. Carrington, 1886).—Daniel R—, æt. 30. Had fever and dysentery last year in Afghanistan. For some weeks ill and shivering; then diarrhœa, dyspnœa, with cough and expectoration.

On admission.—Pyrexia, dulness, and râles at right base behind, no cardiac murmurs, a trace of albumen. Pneumonia extended to left side. Three weeks after admission systolic murmur at apex, and later at base. Weakness and rigidity of left arm, slight facial paralysis.

Autopsy.—Large mass of vegetation on anterior aortic valve, causing ulceration of wall of left ventricle and large mitral orifice. Lungs: right upper lobe tough, solid, rather like a hæmorrhage, but possibly pneumonic; left lower lobe similar; pus in tubes. Liver and spleen soft. Kidneys healthy. Meningitis of base of brain, and of spinal cord; no tubercle; no emboli.

Case 13 (Dr. Pye-Smith, 1887).—Louisa J—, æt. 27. Rheumatic fever and scarlet fever four years ago. Chorea during the last month.

On admission.—Choreic, with soft blowing systolic apex murmur, and diffused beat of heart. Had to be removed to strong room two days later for screaming. Later on was delirious, then violently maniacal, and died exhausted three weeks after admission. Pyrexia imperfectly recorded.

Autopsy.—Old mitral endocarditis, with recent vegetations, one nearly detached. Hæmorrhage at base of left lung. Two infarcts in left kidney. Rather hard spleen.

Case 14 (Dr. Pye-Smith, 1888).—Henry B—, æt. 31. Last year caught cold; never well since. In January had rheumatic fever.

March 12.—Subacute rheumatism, mitral and aortic murmurs. Considerable pyrexia with daily oscillations, except four days, when it was subnormal. Purpuric spots on abdomen, then on chest, face, and all over body; albuminuria; large tender spleen.

April 19th.—Coma.

24th.—Cheyne-Stokes' breathing. Conjunctival hæmorrhage.

Autopsy.—Septic infarction and myocarditis of left ventricle. Old endocarditis of aortic and mitral valves, with recent vegetations on them and in auricle. Pneumonia and pleurisy. Infarcts in spleen probably commencing to suppurate. Kidneys: pinkish-white cortex, with minute petechiæ and small wedge-shaped suppurating infarcts.

Case 15 (Dr. Pye-Smith, 1888).—Richard F—, æt. 38. Rheumatic fever at 16; several attacks of "gout" in foot. Two months' cough, and œdema of feet and eyelids.

May 16th.—Stout and very pale; purpura of feet and legs; double aortic and mitral systolic murmurs. Large liver and spleen.

19th.—Albuminuria. Increasing orthopnœa, râles and rhonchi.

June 6th.—Relief by walking about, but delirious at night. Temperature fell, and he died almost suddenly next morning. Until this time temperature had been pyrexial, with fairly regular oscillations.

Antopsy.—Ulcerative endocarditis of mitral and aortic valves. Caseous bronchial glands, with superficial ulcers on vocal cords and epiglottis. Slightly nutmegged liver. Infarcts in spleen and kidney. Wasted brain, with one wedge-shaped nodule in supra-marginal convolution, due to recent embolus.

Case 16 (Dr. Pye-Smith, 1890).—F. T—, æt. 21. Rheumatic fever 3½ years ago; cough three months; legs swelled six weeks. Pallor, enlarged liver, albuminuria; apparently slight optic neuritis; pyrexial temperature oscillating for the most part between 98·5° and 102° or 103°. Large heart, mitral regurgitant and double aortic murmurs. Death two months after admission.

Autopsy.—Pericardium not inflamed; fifteen ounces of serum. Heart hypertrophied and dilated. Vegetations on two aortic valves, on mitral valve, and in auricle; one chorda ulcerated through. Large congested liver; large soft spleen, with two yellow infarcts. Kidneys large, petechial, with several small infarcts. Petechiæ under mucous membrane of bladder.

Case 17 (Dr. Pavy, 1889).—Kate M—, æt. 22. Rheumatic fever in 1882. Aortic and mitral disease; profound anæmia; retinal hæmorrhages.

Autopsy.—Aortic valves shrunken, thickened, with vegetations on one cusp. Narrow thickened mitral, one chorda broken and knotted into a mass with one adjacent. A mass of granulations on the wall of the auricle, and an ulcer on the mitral valve corresponding to this. Tricuspid stenosed, cusps shrunken and thickened. Œdema of lungs. Nutmeg liver. Infarct in spleen. Kidneys, glomerular and interstitial nephritis, uniformly diffused. A slightly pigmented erythematous eruption on the skin.

Case 18 (Dr. Moxon, 1886).—George W—, æt. 18. Chorea nine years ago, recurring from time to time. No rheumatic fever. Subject to palpitation and irregular breathing.

On admission, June 16th.—Choreic movements, pallor, pains in abdomen and loins, presystolic murmur; no albuminuria.

17th.—Effusion in left knee-joint.

19th.—Left leg numb; left foot white and waxy, with purple patch on dorsum; no pulsation in dorsalis pedis or posterior tibial of left side, and very feeble pulsation in popliteal and femoral; profuse perspiration.

20th.—Right hand cold, and radial pulse indistinct; coldness and numbness extended up the arm at night. Death early on 21st. Temperature high the first three days, falling before death.

Autopsy.—Malignant endocarditis, with emboli of left posterior cerebral and left common iliac arteries. Commencing gangrene of left leg. Brown induration of lungs, with broncho-pneumonia. Infarcts in spleen and kidneys.

GROUP II.

CASE 19. *Gonorrhœa; pulmonary endocarditis; emboli of pulmonary artery; albuminuria; nephritis.*—F. T—, æt. 19, was admitted under Mr. Durham's care on October 7th, 1874.

About four months ago he had gonorrhœa, the inflammatory stage lasting three weeks, and the discharge continuing up to the present time. About six weeks ago he had severe shivering twice a day for a fortnight, terminating in copious sweating.

On September 28th he had sharp pain at the pit of the stomach on taking solid food, accompanied with epigastric tenderness, severe cough, and spitting of blood-streaked sputum.

On admission he had dyspnœa, pain on deep inspiration on the right side, and dulness over the right lung. There was a basic diastolic murmur. Temp. 103°.

On October 10th the following note was made:—The apex-beat is just below the nipple; there is a double murmur over the base of the heart. The right base of the lung is dull, and there is moist crackling with some sibilant rhonchus over both lungs. Pain at the ensiform cartilage, aggravated by deep inspiration. I was asked to see him and noted:—"He appears to have aortic disease. Has also bronchitis of both lungs, and probably pleurisy near the diaphragm on the right side."

He continued to be febrile, the temperature being frequently above 101°. He was often sweating profusely, and had a severe rigor on October 13th. During the latter half of the month he had free sweating, occasional rigors, cough and expectoration, pain in the epigastrium, and a temperature varying between 99° and 103°; sibilant rhonchi in the chest, with râles at the right base.

On November 2nd the urine contained albumen, but no pus; the albumen was increased on the 5th, and he continued with symptoms similar to those above mentioned, with the addition of much pain, tenderness, and fulness in the right loin.

On November 23rd he was much swollen, especially in the face, and the urine contained much albumen and many epithelial casts.

On November 29th the feet were more swollen, and the abdomen contained some fluid.

On December 5th the œdema of the hands and feet was much less.

On December 6th I made the following note:—"Œdema of feet, loins, face; fluid in abdomen. Moist râles over left lung behind; double bruit audible at base. Tongue now moist, fissured, not furred. Urine full of albumen, about three quarters; casts. Pain in side not present now. No sickness. No shivering."

On December 15th he passed 50 oz. of urine.

On December 16th increasing œdema is noticed. He died on the 28th.

Autopsy.—The *lungs*, like all the organs, were very bloodless, just like those of a patient with renal disease. Some parts of them were very œdematous. There was no infarct, nor anything resembling pulmonary apoplexy. The main branches of the pulmonary artery were free, but many of the smaller ones (and even second and third divisions) in each lung contained crumbling masses of white clot, exactly like the vegetations in the pulmonary valves. They were caught upon the forks of the arteries; some of them were loose, others rather firmly adherent to the arterial coats. In the upper part of the left lung there was an extensive but ill-defined patch of grey hepatisation. A connection between this and embolism of the corresponding branches of the artery could not be made out.

The *heart* was not enlarged. On laying open the pulmonary artery it was found that the right anterior valve was covered with a large mass of vegetations, which projected and hung down for some distance. The posterior valve had a considerable part of its edge neatly cut out, just as if it had been done with a pair of scissors. It was not thickened in any way, and had no vegetations on it at this part, although there were some where it joined the right anterior valve. The left anterior valve was perforated with a small hole, also neatly cut out. Evidently these were caused by the pressure of the mass of vegetations. The other valves were healthy.

The *spleen* weighed 6 oz., and was not enlarged, as in ulcerative endocarditis of the left side of the heart. The *kidneys* weighed 21 oz., were very pale and mottled, and evidently in an advanced state of epithelial nephritis. The *bladder, urethra,* &c., appeared healthy.

Case 20. *Endocarditis of aortic and tricuspid valves; empyema; purulent pericarditis.*—Alfred P—, æt. 30, was admitted under me on December 9th, 1888.

Except for an occasional bilious attack, he cannot remember that he has ever been ill. He has been rather a heavy drinker for the last few years. He is a butcher, and says that he caught a bad cold three weeks ago. As this got worse, he had cough, and then pain in the left side on taking a long breath.

We were informed by his medical man, however, that he first saw him three or four days before admission, when he was suffering from "delirium tremens"; that he then spoke of pain in the left side; and two days later this side was examined, and physical signs of fluid discovered. The doctor also then learnt for the first time that three weeks previously he had fallen downstairs, but did not consult anyone for the injury.

On admission.—He is a strong man, in good condition; but his hands are tremulous and his speech thick. Temp. 102·2°; pulse 144; resp. 44.

There were physical signs of liquid (dulness, absence of breath-sounds, ægophony) on the left side, and a needle was inserted in two places below the left scapular angle, but no fluid was drawn off.

The heart's apex could not be distinctly felt; its dulness was not distinguishable from that of the lung. The sounds were normal. The pulse was weak, compressible, and rapid.

During the next few days he was delirious, and resisted examination; but the physical signs appeared to be the same.

On December 15th it is noted that the heart's apex cannot be felt; but the heart seems to be in its normal position, as the sounds are best heard on the left side, in the fifth space internal to the nipple.

On the 17th, however, the præcordial dulness extended to the right of the sternum, and a pericardial rub was heard in the fourth space one inch beyond the right border of the sternum.

On the following day exploration of the left pleura showed a thin purulent fluid, and on December 19th, 16 oz. of thick purulent fluid were removed with the aspirator. I did not attempt to draw more, as I had decided that the chest ought to be incised, and this was done by Mr. Golding-Bird on December 23rd.

He seemed to be relieved by the removal of the pus, but it made no impression upon his temperature, which presented the same features of evening rise and morning fall which had persisted throughout. Diarrhœa was noted on the 21st, and this persisted until January 1st. He was delirious on December 31st and on January 1st. The following day he became drowsy. His respirations increased in frequency; bronchial breathing and loud râles were heard over the right lung; urine and fæces were passed under him; and on January 3rd he died.

The temperature on December 18th ranged from 100° to 102°; during the next five days it was not higher than 100·6°; from the 16th to the 22nd it ranged between 100° and 103°, but was not perfectly regular in its oscillations. From the 23rd December to the day of his death it had a more regular remittent type, being from 99° to 100° in the morning, and 103° in the evening, up to the 29th December, and from 98° to 102° afterwards.

Autopsy.—Left pleura thickened and adherent, very little pus in the cavity. Right pleura normal.

Lungs.—The right lung at the posterior and middle part of the lower lobe, shows a triangular patch of blood-clot, evidently recent infarct, not breaking down. The left lung is tougher than normal, and slightly compressed.

Pus in the smaller tubes of both lungs. Mediastinal glands swollen.

Pericardium contained 1½ pints of thick pus.

Heart of normal size, densely covered with purulent lymph, which seemed but slightly to have affected the subjacent muscle. Mitral and pulmonary valves quite normal. Two aortic cusps were quite healthy, the third had loosely attached to its ventricular surface and edge a smooth, rounded, soft, yellowish-white mass, about the size of a filbert. On the tricuspid there were large irregular soft masses attached, yellow and purulent looking.

Liver.—84 oz. slightly nutmegged. *Spleen.*—14½ oz., soft.

Kidneys.—17½ oz., large and coarse looking, but apparently healthy. Most of the joints were opened; none contained pus.

Case 21. *Tricuspid endocarditis; anæmia; albuminuria; rigors; infarct in lung; pleurisy.*—Elizabeth L—, æt. 17, was admitted into Guy's Hospital under my care on January 17th, 1882, complaining of pains in the back and legs. Her father, mother, one brother, and three sisters are alive and well. One brother, aged three, died of pneumonia, and a sister died at eighteen months, the cause of death unknown to the patient.

The patient cannot remember having any illness herself. She gives no history of rheumatism, or lung affection, or cough, previous to the present illness. She has been ailing for about seven months, and in July her menses stopped. In September she had some medicine, which was followed by a little flow, but she has since then not seen anything at all. About a month ago she first noticed pains in the back and legs, and she could not kneel because her legs were stiff. These pains were getting worse, when she came to the hospital as an out-patient, and was admitted.

On admission.—She lies on her side, looking half-starved; very thin; skin dry. She has brown hair, grey eyes, pupils widely dilated, conjunctivæ clear and pearly. Teeth white and even. Some slight dulness at left apex; vocal resonance rather more marked on right than left side. Vocal fremitus the same on both sides.

Heart's apex in fifth space, about two inches below, and one inch internal to the nipple line. At the apex the heart sounds are normal; at the base there is a loud systolic bruit over the second left interspace; it is loudest near the sternum, presumably of pulmonary origin. Tongue is dry, glazed, somewhat furred in the centre. Hepatic dulness not increased. Spleen normal. Bowels regular.

Urine pale, of sp. gr. 1010. No albumen or sugar. Pulse 96; temp. 98·4°; evening temp. 103·4°.

January 18th.—Morning temp. (8 a.m.) 102°, (11 a.m.) 99·6°; resp. 29.

19th.—Eats but little; has much thirst. Morning temp. 102·6°; pulse 130; evening temp. 105°.

20th.—To-day takes food well. Pulse 120, soft; morning temp. 101·2°; evening temp. 103·4°.

21st.—Morning temp. 101·6°; evening temp. 104·2°.

22nd.—Morning temp. 104·4°; evening temp. 104°.

23rd.—Temperature rose to 106·6°, but fell spontaneously in three hours to 101·6°; evening temp. 100·6°.

24th.—Bowels were opened last night, the motions being clay-coloured and scybalous. There is crepitation at both bases. At 11.45 a.m. she had a rigor; the temperature in the mouth was 106·4°, in the right axilla 106·4°, and in the left axilla 107·6°; pulse 170. At 12.15 she had 10 grains of quinine; cold sponging for fifteen minutes. From 12.40 to 12·50 she was in a bath of 90°, reduced to 77°. At 1 p.m. had another rigor. After the bath the temperature was in the mouth, 99·8°, in axilla 102·2°. At 2.20 the temperature in the axilla was again 105·2°. Ten grains of quinine were again given. At 3.15 the temperature was 106°; she was again placed in bath, and at 6 p.m. the temperature was 101·2°. She was then deaf; the tongue moist, red with white patches. Bowels open; the motions as before. Profuse sweating; alæ nasi working.

25th.—Comfortable; no pain; tongue as before. Abdomen distended. Pulmonary bruit very loud. Patient very weak and will not answer questions. Morning temp. 95·5°; evening temp. 101·2°.

26th.—At 2 a m. the temperature rose to 105·8°. She looks ill, with livid face. Resp. 40; pulse too weak and rapid to count. Quinine 20 grains.

Temperature was reduced by sponging to 100·4° in the mouth, 102·8 in the axilla. Spleen can be felt descending during inspiration when hand is placed deeply under the ribs. The temperature fell later to 98·4°, and rose in the evening to 106·2°.

27th.—Morning temp. below 97°. Not deaf; no headache; bowels acted once in the night and once this morning. Evening temp. 106°.

28th.—She continues sensible; talks quietly; face sometimes pale, sometimes livid. Breathing laboured, gasping. Increased area of cardiac dulness; pulmonary bruit as before. No pains. Pulse small, 120; skin hot and dry. Tongue moist, with small white patches of fur. Morning temp. 102°; evening temp. 104°.

29th.—Bowels loose. Morning temp. 103°; evening temp. 104·2°.

30th.—Urine high coloured, of sp. gr. 1025; loaded with albumen and blood. Great thirst. Morning temp. 102·7°; evening temp. 104°. Ordered quinine 20 gr., linctus opiatus, p. r. n.; milk, beef-tea, two eggs; brandy 8 oz.

31st.—Urine as before; increased splenic dulness; crepitations at bases, with deficient breath sounds. Morning temp. 97·6°; evening temp. 103·2°.

February 1st.—Urine contains blood and three-eighths albumen. Has much thirst; cannot take beef-tea; vomited during the night. She has pain in the right elbow-joint, which is not tender to pressure, but painful when the forearm is flexed or rotated. Morning temp. 102°; evening temp. 105·2°.

2nd.—Much retching. Took 2½ pints of milk during the night. Pain in right elbow; pain at lower part of right side of chest; a poultice ordered.

3rd.—Urine of sp. gr. 1016; albumen one eighth; no blood. Splenic dulness as before; abdomen distended, tympanitic; very slight cough, no sputum.

4th.—Dry cough; no pain; bowels opened three times. Lies on left side with knees drawn up. Abdomen soft and full; no spots. In the afternoon face paler; breathing noisy; pulse increasing and very compressible. Wanders now and then. No dulness over lungs, except at extreme right base. Breath sounds rather exaggerated. Pulse 176; resp. 60. Temperature oscillates between 98° and 105°.

5th.—Right chest nearly full of crepitations; left side supplemental breathing. The face has more colour; the lips are not so pale. She does not take her food so well. Passed urine under her.

6th.—Bowels relaxed, moved five times this morning; everything passed under her. This afternoon fall of temperature to 98·8°; much sweating and blueness of lips. Both chests full of crepitations. Enema Opii given in the evening.

7th.—No motions since the enema. Is very pale.

8th.—Three motions to-day; mind wandering.

9th.—Two motions to-day; increased delirium.

10th.—Very thirsty; continued delirium. Bowels opened six times in twenty-four hours. Resp. 38. Slightly better colour.

11th.—Noisy, singing and talking all last night, and never slept. Pulse 130; resp. 34. Loose motions three times in the day.

12th.—Continued delirium; face yellowish; laborious breathing; holding breath at end of inspiration, and then expiring forcibly. Temp. 99°; pulse 128°.

13th.—For some few days the epigastric region has been curiosly prominent to the left of the middle line, and there is distinct dulness. To palpation it is elastic, not very tense, giving the idea of a cyst rather loosely filled, not nodular. There is decided pain on pressure, most severe at the site of the prominence. At my request, Mr. Jacobson saw the patient, and, after administration of ether, punctured the tumour with a small trocar, but drew off nothing but blood, which showed under the microscope a large proportion of white cells, not, however, amounting to leucocythæmia. This operation was done antiseptically.

14th. — The blood was examined microscopically, and found to contain many white cells, but not a quantity sufficient to constitute leucocythæmia. In the afternoon she became very noisy, screaming apparently with pain; and she died at 8.30 p.m.

Autopsy.—There was recent lymph over the whole of the right lung, forming a thick layer upon its posterior surface. Probably this had started from the infarctions.

Both *lungs* were very heavy, and massive, but smooth on section, and rather tough than soft. There was œdema of the upper lobes. In the right lower lobe there was a wedge-shaped infarct, with a grey border. The artery going to this was plugged with thrombus, softening down into a puriform liquid.

There was adhesion of the *pericardium* over the left apex. The *heart* weighed 9 oz.; both right and left cavities were rather dilated. All the valves were healthy, except the tricuspid, the anterior flap of which had a large mass of smooth vegetations hanging from it. Some of the chordæ were probably torn through. There was a minute aperture, leading through the weak spot of the septum into the left ventricle.

The *liver* weighed 70 oz., was congested and soft, probably fatty. In the left lobe was the puncture made by the trocar, forming a blood-stained track an inch long. The *spleen* 7 oz., and the *kidneys* 11½ oz., were healthy.

The femoral veins were healthy. No cause of blood infection could be traced.

Case 22. *Vaginitis; pulmonary endocarditis; delirium; diarrhœa; peritonitis; infarct in lung.*—Elizabeth J—, æt. 3, was admitted into the Evelina Hospital on November 18th, 1881.

The parents are healthy; the child is one of three living; one child died of bronchitis, another from diarrhœa, and a third from an accident. There is no history of phthisis. The house they live in is said to be damp. The child has had varicella, but never measles, nor scarlet fever, nor pertussis. There is no evidence of congenital syphilis. She was brought here as an out-patient three weeks ago, with cough and some vaginitis. She improved, but still remained feverish; otherwise fairly well, till three days ago, when she shivered; the skin became very hot and dry. Last night she woke up with another rigor; has not coughed or complained of cold.

On admission.—Resp. 56; pulse 144; temp. 104·6°. She is very pale and weak, but fairly well nourished; mucous membranes rather anæmic; no cutaneous eruption. The tongue is moist, the dorsum covered with thin white fur. She is thirsty, takes much milk; the bowels were open twice last night, the motions being loose, and of light yellow colour.

The impulse of the heart is diffused, difficult to localise, but apparently not displaced; no increase of cardiac dulness; the action rapid but regular. A loud systolic bruit is audible over the whole chest, but of greatest intensity over the pulmonary cartilage, where it is rather higher pitched than elsewhere, and can be markedly intensified by pressure with the stethoscope.

Resonance is somewhat deficient over the apex of the left lung in front; otherwise normal.

Rather distended abdomen, but no tenderness or gurgling; spleen not felt; no spots; much general hyperæsthesia. Temp. 104° to 105°; is quite conscious; no delirium.

On the evening of admission, at 10.30, the temperature rising to 105·2°, she was bathed at 90° to 95°. The pulse was intermitting for ten minutes after the bath. She then slept fairly well, but sweated much, and started in her sleep, without, however, talking. She took milk and barley water well. The temperature after the bath was 102·2°, and at 12 midnight was 99·2°. The temperature rose during the morning of November 19th to 103·8°, and fell again at noon to 100·2°, rising again at noon to 103·6°. During the day she had pain in the head, sweated much all over the body; objected to the light.

November 20th.—Has slept fairly well, but wandered much; slight sweat; pain in the stomach, and also at the heart, after 6 a.m. Temperature remained above 102·6° till 6, then fell to 99° at noon. She was fretful all day, with pain in left side, in stomach, and in heart; asks for all she wants. Three loose motions. The temperature rose in the afternoon to 102·8°, and remained about 102° till the evening of the 21st.

21st.—Slept well all night; wanders a little during sleep; took milk well; complained of headache. During the day was fretful; slight sweat; legs drawn up, but no complaint of pain. Temperature fell at midnight to 97·2°.

22nd.—Slept well last night. No pain. No loose motions.

23rd.—The murmur is loudly audible under the left clavicle, distinctly but not so loudly at the left apex; loudly behind, both at upper and lower parts of chest. At the pulmonary cartilage the second sound is very loud and rather lengthened; at the sternum, and to the right of it, there is very distinct reduplication.

25th.—During the last three days she has been fretful; restless at night; two or more loose motions daily; has taken milk well. The temperature has been oscillating between 99° and 101°. Last night it fell to 98°, rose at 3 a.m. to 103·5°, and fell at 9 a.m. to 97·2°, without any shivering being remarked. During the day the temperature again rose to 101°. The nurse's record during the next few days is very similar: fretfulness; fairly good sleep; food taken well; bowels mostly confined; temperature oscillating decidedly, often rising and falling a distance of three degrees twice in the twenty-four hours.

On November 27th she was ordered Ferri et Ammon. Citratis, 3 gr., t. d. s.

There was no essential alteration until December 6th. The temperature rose to 102° on the 29th November, remained for some days oscillating between 99° and 102°, and rose to 103·6° on December 5th. She seemed about this time to be more fretful, and had more abdominal pain.

December 6th.—Had a bad night, was restless, and evidently had great pain in the abdomen; she vomited once, this morning just before food, the vomit consisting apparently of curdled milk; pulse small and thready; anxious expression and pallor even more extreme than yesterday; tongue moist and clean; extremities cold; abdomen still distended, but not tympanitic; cough more frequent; lung sounds normal. The temperature to-day was mostly between 102°

and 103°, and at 9 p.m. was 102°. The bowels have not acted to-day.

7th.—Child remained in much the same state until last evening, when the breathing became distressed; she was much weaker and very restless, but sensible. She had also much pain in the stomach, and had four loose motions. She died with struggling about 4 a.m. The temperature had previously fallen, and it was 98·8° one hour before death.

Autopsy.—The *lungs* pale and healthy, except for the presence of a hæmorrhagic infarct in the left lower lobe; no pleuritic adhesions or effusion; no enlargement of the bronchial glands.

Heart.—The pulmonary sigmoid valves presented a mass of vegetations, nearly as big as the top of the little finger, almost entirely filling the lumen of the pulmonary artery. The other valves were healthy, and the heart in other respects presented nothing abnormal.

Abdomen contained an excess of peritoneal fluid; some peritonitis over the spleen, but none elsewhere; no ulceration or anything abnormal in the intestines; the mesenteric glands healthy. *Liver* enlarged; weight 25 oz.; pale, but not fatty. *Spleen* 5 oz. weight; congested; no infarcts. *Kidneys.*—Capsule easily detached; substance pale, but apparently healthy; no infarcts.

Case 23 (Dr. Pye-Smith, 1886; see 'Path Trans.,' vol. xxxviii, 1887, p. 114).—Charles L—, æt. 39. No previous illness known till four months ago; then attacks of dizziness assumed finally the character of rigors every one, two, or three days. Very anæmic, œdema of legs, spleen enlarged, pulmonary basic murmur. Albuminuria. Increasing anæmia; red corpuscles only 30 per cent. of normal. Rigors; murmur at pulmonary base becomes double; diarrhœa. Death fifteen days after admission.

Autopsy.—Extensive disease (ulceration, perforation, and partially calcareous vegetation) of pulmonary valves. Aortic and mitral valves slightly thickened. Lungs anæmic, no emboli or infarcts. No infarcts in other organs.

Case 24 (Dr. Shaw, 1890).—William M—, æt. 16. Was not thought to be ill until March 30th. Had successively giddiness, rigors, pains, and diarrhœa; went to bed, and enteric fever was suspected. On April 8th was pale, with hectic flush, signs of fluid at right base, optic discs healthy, heart sounds normal. Rigors again, loose yellow motions. Temp. 100° to 102° in the morning, 103° to 105° in the evening. Delirium and coma. Death on 16th.

Autopsy.—One or two healing sores, as if from injuries, on the hands. Heart: tricuspid valve alone diseased, presenting a large mass of decolourised clot, with ulceration, perforation of cusp, and detachment of chordæ. Four pints of blood-stained serum in right pleura, one pint in left; both lungs compressed; about ten suppurating infarcts in left lung. Large spleen, several infarcts, yellow but not breaking down. A few pin-points of suppuration on one kidney as if from infarcts.

Case 25 (Mr. Davies-Colley, 1888).—Donald P—, æt. 58. Ill four weeks, unable to pass water; large prostate; albumen, pus, and casts in urine. Day after admission left arm paralysed and stiff. Died following day.

Autopsy.—Heart: a small embolus in left coronary artery, and a suppurating infarct in the wall of the heart. Immediately above the left cusp of the mitral a nodule with a tear of the endocardium. A vegetation on one of the pulmonary valves. Œdematous lungs, with pus in the tubes. Points of suppuration in liver. Pyæmic infarcts in kidney. Suppuration of prostate and of prostatic veins. Brain: a partially decolourised clot in a branch of the right middle cerebral artery; softening and petechiæ of corresponding portion of brain. Another clot in the right anterior cerebral artery, with consequent softening. Suppuration of thyroid body.

Case 26 (Mr. Golding-Bird, 1885).—William R—, æt. 46. A hard drinker. Stricture, retention, and extravasation of urine. Incisions. Abscess formed in hypogastric region. Diarrhœa two days after admission, continued till death, eleven days later. No note of condition of heart. Moderate pyrexia throughout.

Autopsy.—Two aortic valves adherent, with a small ulcer on one of them. Lungs emphysematous; one hæmorrhagic infarct with pleurisy on the surface, and suppurating in the centre. Small firm spleen; congested kidneys; suppuration of wall of bladder and urethra.

Case 27 (Mr. Davies-Colley, 1888).—Thomas McG—, æt. 65. Gonorrhœa three times, stricture, cystitis four years, perineal abscess, pyrexia. Sudden death.

Autopsy.—Large heart; mitral stenosis, with a soft smooth vegetation, as large as a chestnut on the auricular surface of the valve. Œdematous lungs, thirty ounces of serum in left pleura. Small pulpy spleen. Kidneys: cortex deeply scarred by old ascending nephritis, and on section numerous punctiform red spots visible in the cortex. Cystitis.

Group III.

Case 28. *Mitral endocarditis; right hemiplegia; convulsions; anæmia; œdema of lungs.*—Paulina C—, æt. 28, was admitted under my care, May 2nd, 1885.

She had diphtheria at eight years of age, and rheumatism at sixteen; since then has been well until two years last Christmas, when she had severe bronchitis. Her last child was born just after that, and she has not been strong since. She has had to give up work two months. She has during two years suffered various symptoms, a sensation in her throat as if it were going to burst, pains shooting down the left arm, loss of power in the calves of the legs, giddiness and headache.

For two months she has had gastric pains, vomiting, continued cough, and the last month swelling of the legs.

On admission.—Anæmic, breathing rapidly; œdema of both feet; pulse 88; temp. 99·4°. Impulse of heart in fifth space, 1½ inches below nipple; dulness limited by fourth rib, left border of sternum, and nipple line. Diastolic thrill felt over precordial area. Cantering rhythm, with systolic and mid-diastolic murmur. In second left space a systolic murmur; behind also a systolic murmur. In the third interspace a rough presystolic murmur. Liver enlarged. Urine normal. Sputa streaked with blood, but no marked physical signs in the chest.

On May 7th, at 5.45 p.m., while sitting up in bed, she suddenly became quiet, and unable to answer any question; she then became unconscious, and remained so a few minutes. She was found paralysed in the right arm, face, and leg, and unable to speak at all, though she tried.

At 7 p.m. she had recovered some movements in the limbs, and could answer some questions, but very slowly indeed. At 10.30 she suddenly developed violent choreic movements in the right limbs, neck, and face, and a little twitching at the left angle of the mouth. At 11 p.m. she could speak slowly, and the power of the right arm and leg was greater.

On the 8th the right arm and leg were only weak. Dr. Pitt notes, "She could apparently speak correctly, except she called her left arm, &c., the right. She could write from dictation or from a book, but could not otherwise. She had therefore some agraphia and aphasia, limited to voluntary action."

On the 11th it is noted sensation is not affected, but on the 12th she had a feeling of numbness in the left leg.

May 15th.—Apex sounds are now systolic and diastolic. Tubular breathing at the apex of the right lung.

On May 19th she had an offensive vaginal discharge, and on the 28th Dr. Horrocks reported a fleshy mass adherent to the fundus of the uterus, and advised its removal. This was done by Dr. Horrocks on June 2nd, and the vaginal discharge ceased.

On June 12th she was up for a short time.

On June 21st she had more cough and dyspnœa, and spat up some blood, and the following day examination showed that there was deficient entry of air with râles at the bases. The second murmur is sometimes presystolic, at others mid-diastolic. She is anæmic, and perspires profusely.

On July 4th there was much œdema, more spitting of blood, increased râles over the back, and great rapidity of the heart's action. She got rapidly worse, and died July 8th.

Autopsy.—Brain 41 oz., healthy; no trace of softening or of embolism in any of the arteries.

Lungs.—Old fibroid adhesions on right side and recent lymph over left side, most marked at the base, where it was as thick as blotting-paper over a considerable extent. The lungs were marked examples of brown induration. They were very firm, and nearly airless throughout; very œdematous, and the smaller tubes full of muco-pus. The lower lobe of the left lung was quite airless, and parts sank in water. It was firmer and harder than the rest, more œdematous, and the tubes contained much more pus. It appeared to be an acute pneumonia supervening on a chronic change. There was an exactly similar condition at the right apex, extending two inches down. The branches of the pulmonary artery were much thickened throughout. The main trunk was quite as thick as the aorta.

Pericardium healthy.

Heart 15 oz. The left ventricle was greatly dilated and much hypertrophied. Both sides were full of post-mortem clot. The muscle was good throughout, but rather pale on the right side. The left auricle was dilated and hypertrophied, the cavities on the right side dilated and somewhat thickened. Chordæ tendineæ nearly all completely detached. These and the mitral cusps were completely covered with recent granulations. The endocardium of the left auricle was considerably thickened, white and opaque, showing that there had been mitral regurgitation. One small patch of calcareous atheroma in the aorta just above the valves; otherwise the vessel was good throughout.

Stomach much dilated and full of light yellow fluid. *Liver* 61 oz., surface smooth, moderately nutmegged. Ducts patent, but the main duct was small.

Spleen 12 oz., very firm. *Kidneys* 13 oz., somewhat enlarged, contained a few cysts. A few minute punctiform ecchymoses in the cortex, and the surface generally injected. With these exceptions the structure looked healthy.

Case 29 (Dr. Carrington, 1885–86).—Violet G—, æt. 22. Never had rheumatic or scarlet fever, but five years ago was told she had heart disease. Ill since then. Admitted with cough, pale and dusky face, mitral regurgitant murmur rather musical than rough, no albuminuria. Later, double aortic, and diastolic and præsystolic mitral murmurs, enlarged spleen, face and feet swollen, high temperature, urine albuminous, face and feet swollen, œdema of bases of lungs, coma and death.

Autopsy.—Fungating endocarditis of aortic and mitral valves; the aortic valves thickened; extreme stenosis of mitral valve. Nutmeg liver. Very large, firm spleen, with three large infarcts. Kidneys 19½ oz. Red-pink mottled with yellowish-white. Punctiform hæmorrhages, swollen cortex, one infarct. Brain: small embolism in left middle cerebral artery; no softening. Brown induration of lungs.

Case 30 (Dr. Pye-Smith, 1885).—Ellen A—, æt. 42. Admitted for dyspnœa and œdema of legs; aortic and mitral disease, large liver and spleen; albuminuria. Increasing dyspnœa, orthopnœa, wasting. Pyrexia not recorded.

Autopsy.—Cord-like pericardial adhesions; each aortic valve perforated and occupied by fungating granulations; stenosis of mitral orifice, with ulceration of one valve, and rupture of one chorda. Nutmeg liver. Infarcts in kidneys, and one in the left suprarenal capsule. Lungs indurated.

Case 31 (Dr. Moxon, 1885).—Arthur E—, æt 30. Three attacks of rheumatic fever. In hospital two years ago with cardiac disease. Double aortic murmur, large heart, large liver, large tender spleen, albuminuria, with casts. Œdema of legs, with erythematous and purpuric rash on legs, hands, and face. Temperature 103°. Died day after admission.

Autopsy.—Adherent ecchymosed pericardium. Ulcerative endocarditis of aortic and mitral valves. Pneumonia of right lung; both œdematous. Large cirrhosed liver; large spleen; kidneys 28 oz., subacute parenchymatous nephritis. Brain healthy.

Case 32 (Dr. Pye-Smith, 1884–85).—Alice H—, æt. 39. No history of rheumatism; cardiac symptoms seventeen months. Rather pale; dropsy and purpura. Large heart, with mitral murmur. Albuminuria. Erysipelas of face recurring at intervals of weeks; pericarditis; increasing cyanosis; death.

Autopsy.—Pericarditis, with 8 oz. of serum and recent lymph; constricted mitral orifice with ulcerated chordæ, and vegetations in left auricle. Tricuspid thickened. Indurated lungs, with old phthisis at one apex. Nutmeg liver. Infarcts of spleen and kidneys. Fatty epithelium in renal tubules. Brain healthy; a firm decolourised embolus at bifurcation of right common carotid.

Case 33 (Dr. Pavy, 1887).—John B—, æt. 8. History vague and uncertain. Admitted drowsy, vomiting, with dropsy, large heart, mitral murmurs, clubbed fingers, and large liver. Increasing dyspnœa and drowsiness. Death a few days after admission.

Autopsy.—Calcareous change in one aortic cusp, the others almost entirely destroyed, with clumps of vegetations clinging to the remains. Beads on mitral. Congested lungs. Infarcts in spleen and kidneys.

Case 34 (Dr. Pavy, 1888).—William M—, æt. 21. Rheumatic fever twice at thirteen and sixteen. Recently cardiac pain on exertion; then a temporary paraplegia, from which he seems to have recovered. January 15th, orthopnœa, drowsy, slight ascites, enlarged liver, large heart, with mitral and aortic murmurs, pyrexia very slight. Died rather suddenly on January 22nd.

Autopsy.—Ulcerative endocarditis of mitral valve, with ruptured chordæ infecting auricle and aortic valve. Thickened and retroverted aortic valves. Indurated lungs. Nutmeg liver. Infarcts in spleen and kidney. Spinal cord appeared healthy.

Case 35 (Dr. Pavy, 1888).—Elizabeth S—, æt. 25. Rheumatic fever six times since thirteen. Cardiac symptoms twelve months. Pale, flabby, with œdema of feet, petechiæ on arms, legs, chest, and back. To-and-fro basic and presystolic apex murmurs; bronchitis; albuminuria; enlarged liver; vomiting; diarrhœa. Died a week after admission. Record of temperature missing.

Autopsy.—Short thick incompetent aortic valves; mitral valves thickened, numerous vegetations. Tough liver and spleen. Petechiæ in kidneys and glomerular nephritis. No micro-organisms were found in the vegetations, but the sections did not stain well (Dr. Pitt).

CASE 36 (Dr. Hale White, 1889).—Thomas B—, æt. 19. Rheumatic fever three years ago. Systolic mitral, to-and-fro aortic; and pulmonary murmurs, heart irregular, dyspnœa, œdema, albuminuria. Record of temperature missing. Death eight weeks after admission.

Autopsy.—Thickened aortic valves; thickened mitral, with ruptured chordæ tendinea. Lung indurated and pneumonic. Nutmeg liver. Petechial kidneys.

CASE 37 (Dr. Moxon, 1886).—George M—, æt. 19. Chorea nine or ten years ago, and repeated attacks since; then cardiac disease.

Autopsy.—Ecchymoses under pericardium. Mitral orifice narrow; both cusps and tips of musculi papillares thickened. Much fungating endocarditis of one cusp, with perforation of cusp, and rupture of chordæ. Granulations on the tricuspid. Induration of lungs and early broncho-pneumonia. Subpleural ecchymoses. Infarcts in spleen and one kidney. Brain: ecchymosis over right supra-marginal convolution; a decolourised clot in the left posterior cerebral artery. No softening.

CASE 38 (Dr. Pye-Smith, 1886).—Leonard G—, æt. 39, Acute rheumatism eight years ago, cardiac disease since. Died day after admission.

Autopsy.—Large heart with adherent pericardium. Aortic valves thickened and everted. Mitral: anterior cusp thickened, chordæ ruptured, thickened, and twisted, but without granulations. Red hepatisation of both lungs, with early pleurisy. Nutmeg liver. Large firm spleen, with three recent infarcts. Large congested flabby but otherwise normal kidneys.

CASE 39 (Dr. Goodhart, 1887).—Martha S—, æt. 58. Appears to have had some uterine disease. Admitted with heart disease, albuminuria, delirium, and died in a week.

Autopsy.—A patch of ulceration about the size of a shilling, partly on the valve, partly on the auricular wall. Lungs: marginal emphysema; pus in bronchi and bronchioles. Other organs practically healthy. Uterus congested.

Case 40 (Dr. Pye-Smith, 1890).—George T—, æt. 33. Inflammation of lungs as a child; gonorrhœa three years ago. Four months ago fainted and was ill five weeks. Ten days pain in left chest, and dyspnœa, which has increased. Now sallow, half icteric. Dyspnœa; large liver; dulness and râles at both bases. Aortic and mitral disease. Albuminuria. Died suddenly three days after admission.

Autopsy.—Vegetations on aortic and mitral valves; aneurysm of one of the mitral cusps. Fluid in both pleuræ; congested lungs; large spleen; infarcts in kidneys.

Group IV.

Case 41. *Mitral endocarditis; albuminuria; convulsions; cerebral hæmorrhage from aneurysm; infarcts in spleen; nephritis.*—George B—, æt. 31, was admitted on February 17th, 1885, under Dr. Wilks's care, and subsequently came under me. He had gastric fever at eighteen years of age, and rheumatic fever six years ago. He has never been strong since then. He has suffered from winter cough, and he gave up work on October 30th.

On admission.—He is anæmic and thin. He suffers from dyspnœa and palpitation. The heart's impulse is in the sixth space, half an inch external to the nipple. There is at the apex a systolic murmur and a doubtful diastolic murmur. At the base is heard a to-and-fro aortic murmur. The pulse has a water-hammer character. There are a few râles at the back of the chest. The hepatic dulness reaches an inch below the ribs. The spleen cannot be felt. The urine has a dusky tint, contains albumen, and responds to the guaiacum test for blood. There are no casts.

He was ordered digitalis, with ammonio-citrate of iron and carbonate of ammonium.

On February 28th he was allowed to be up. On March 29th he complained of pain down the back of the right leg, which subsided in a few days. Albuminuria was noted as present on March 17th, and towards the end of the month he suffered from headache. On May 1st he suffered from marked giddiness, and on the night of the second he had a fit of general convulsions. He had a second fit at 1.30 and another at 2 on the 3rd. The convulsions began on the left side of the face; as he recovered, the eyes were open, and directed upwards to the left. He had six more fits on this day, remaining unconscious the greater part of the time between them, and passing his urine into the bed. He gradually recovered his senses.

He passed a good night, but on May 4th was twice sick. The urine contained blood and albumen. On May 5th it is noted that his temperature has been nearly normal the last two days. He continued much the same during the next week. He had no return of fits nor of sickness, and he improved in appetite, but he was troubled with cough.

The urine contained albumen, but no blood. On the evening of the 15th he was sick, but he had imprudently eaten some cherries. He died on the 17th.

Autopsy (by Dr. Goodhart).—*Brain.*—A quantity of extravasated blood was found extending over the surface of the base, and some distributed over the motor area on the right side of the cortex. Its distribution here, however, was peculiar. The veins of the cortex were full of blood, and extravasation had taken place in their course. The cortex of the motor convolutions was minutely ecchymosed all over. At the base the right middle cerebral seemed thick and diseased, and on further examination a circular or oval clot was turned out of the brain substance just below the surface. There was a very extensive extravasation of fluid into the brain substance. It did not involve the internal capsule or the large ganglia, the outer part of the lenticular nucleus being quite free. Ultimately a large aneurysm was found measuring about one inch by three quarters of an inch. It was not quite clear where it had ruptured, the aneurysmal sac being only about half complete; that is to say, the one half (about) of the sac was like arterial coat, the other was quite a recent clot. Dr. Goodhart thought, therefore, that the aneurysm must have enlarged rapidly lately and then ruptured, the original sac being spread out as it was found in this manner. The other vessels were healthy, and no plug could be found.

Lungs and *pericardium* healthy.

Heart.—Weighed 21½ oz. Mitral valve thick and small; its tendinous cords much thickened. Many were ulcerated through leaving free projecting ends, and the whole valve and the cords were fringed and sanded with vegetations of all kinds. The aortic valves were thick. The auricular wall had a thick carpet of vegetations on its posterior surface.

Liver.—63 oz., rather soft. *Spleen.*—15½ oz., one recent infarct, one old. *Kidneys.*—13½ oz., fawn-coloured on the surface, with ecchymotic patches in many places. Minute examination showed much speckled inflammatory material.

CASE 42. *Aortic endocarditis; pyrexia; delirium; left hemiplegia; softening of the right lenticular nucleus.*—Emma B—, æt. 38, was admitted under my care on January 9th, 1889.

The only history to be obtained is to the effect that she has been addicted to drink, that she has been ill for nine months, and that she has lost flesh rapidly the last fortnight. Two days ago she is said to have fallen off a chair.

On admission.—She is emaciated, and looks old for her years. She is in a condition approaching collapse, drowsy and rather incoherent. She complained loudly on being touched, but there was no particular tenderness about any joint. She used her limbs uniformly though feebly. There was a loud diastolic musical murmur, heard all over the chest, and at the apex a soft systolic bruit. Râles on both sides of the chest behind. Temp. 101·6°.

January 10th.—A more complete examination shows a diffused cardiac impulse, three inches in diameter, reaching out to the nipple line; a loud musical diastolic murmur, loudest in the aortic area; a soft blowing systolic murmur at the apex. P. 116, feeble and rather splashing; the arteries seem good.

Respirations are 30 to 40, shallow. Cough is frequent but feeble; no expectoration. There is dulness at the right base up to the eighth rib, with loud consonating râles, rhonchi, bronchophony, and a doubtful rub. At the right base there is an extensive dulness, with râles and rhonchi.

At 12.30 there was marked left hemiplegia, the face, arm, and leg being involved; the arm completely paralysed, the leg less so. The patient spoke thickly, but was not aphasic. Knee-jerk and elbow-jerk were more marked on the left side, and on the same side patellar and knee-clonus were present. Hyperæsthesia less than on admission. Optic discs normal. Abdomen flaccid. Spleen not felt. Liver dulness reaches from the seventh rib to within two inches of the umbilicus.

Urine, sp. gr. 1010, acid, clear, no albumen or blood, but many casts and granular epithelial cells are found under the microscope.

January 12th.—Low delirium; she throws off the bedclothes, and tries to get out of bed. Breathing more rapid and stertorous.

14th.—P. 130. Impulse of heart more diffused and fluttering. The diastolic murmur has lost its musical character. Breath-sounds more feeble, râles less loud. Temp. 99°.

15th.—Noisy and delirious; semi-conscious; when roused, complains of pain in various parts, and is generally hyperæsthetic. The right knee-jerk is pronounced, and ankle-clonus is present on this side also. At 4 p.m. no bruit could be heard, only a rapid tic-tac. Dulness and bronchial breathing at right base; dulness with doubtful bronchial breathing at left base. She died at 9 a.m. on the 16th.

16th.—After a draught last night of potassium bromide 20 grains, and chloral 7 grains, the delirium subsided; she died quietly at 9 a.m. this morning.

Autopsy.—The *brain* weighed 43 oz. Membranes rather milky, some wasting of convolutions, vessels good.

In the right lenticular nucleus at its lower part, just outside the internal capsule, and one inch behind the genu, was a patch of white softening about the extent of a shilling in horizontal area. No embolism of vessels could be found. The rest of the brain was firm.

Lungs.—Emphysema of left upper lobe, much lobular pneumonia in the left lower lobe. Lobular pneumonia, less marked in the right lower lobe. The bronchial tubes were inflamed, and contained pus. Recent pleurisy at right base.

Heart 11 oz. Much colourless tough post-mortem clot in the ventricles. Fungating endocarditis on ventricular surface of all the aortic cusps. The anterior cusp had a large perforation in it, while the left posterior had a large aneurysmal bulging, which would have soon led to a perforation. The right posterior cusp was also heaped up with vegetations. The vegetations were remarkably colourless, and showed little signs of infection beyond the endocardium immediately beneath the attachment of the cusps. The other valves were quite healthy. No evidences of regurgitation through the mitral. Left ventricle not noticeably dilated. Cardiac muscle and vessels good. *Stomach* perhaps a little congested. *Spleen* 6 oz., normal.

Kidneys 7½ oz., small. A little wasting of the cortex in places, and one or two small cysts, but the capsule peeled well, and the surface was quite smooth.

Case 43 (Dr. Hale White, 1886).—Elizabeth H—, æt. 13. Chorea three years ago; heart diseased one year after. Five weeks ago began to shiver, then had pain in the joints.

On admission.—Joint pains, pyrexia, mitral regurgitant murmur. Nine or ten days after admission screamed and had convulsions; two hours later another scream and convulsions, and continued unconscious. She had a third convulsion after six hours, and never rallied.

Autopsy.—Adherent pericardium; mitral endocarditis, with several ruptured chordæ. Recent infarcts in spleen. Brain: recently extravasated blood in left lateral ventricle tearing away large ganglia; outside this an older clot in connection with a vessel in the Sylvian fissure, on which was a fusiform aneurysm.

Case 44 (Dr. Hale White, 1887).—Jane L—, æt. 23. Rheumatic fever twice. Hæmaturia for three months.

On admission.—Dyspnœa, palpitation; mitral murmurs; blood and albumen in urine; enlarged tender spleen. Delirious the following day. Three days before death had a fit, another in the night and two the next day. Abdominal pain and tenderness; increasing râles and rhonchi. Temperature only slightly raised.

Autopsy.—Masses of vegetations on anterior flap of mitral valve, and large masses on wall of auricle. A large, soft, flabby spleen, with two large infarcts. Many infarcts in kidneys, in which organs the microscope showed cirrhosis and wasted glomeruli. Brain: a doubtful minute hæmorrhage in left corpus striatum.

Case 45 (Dr. Goodhart, 1887).—Emma S—, æt. 12. Rheumatic fever last year and the year before. Admitted for pain in the joints and pyrexia. Rigors the day before and two days before that. Mitral murmur; continued pyrexia. Fifteen days after admission hæmoptysis, and again the next day. Two days later a fit, with twitching of right eyelids and face, coma, stertorous breathing, and death in ten and a half hours.

Autopsy.—Adherent pericardium; mitral and tricuspid orifices dilated; vegetations on margin of mitral valve, and patch of endocarditis in left auricle. Soft spleen with one infarct; swollen kidneys with one infarct. Brain: blood in the pia mater; left frontal lobe full of blood, old and recent. No aneurysms, emboli, or rupture of vessels found.

Case 46 (Dr. Pitt, 1889).—M. C—, æt. 43. Had a fit in the street, followed by left hemiplegia; albuminuria. Iodide of potassium produced on extensive rash, for which she came in eight days after the fit. Double aortic murmur; albuminuria, moderate pyrexia. Died five days later.

Autopsy.—Vegetations on segments of aortic valves, orifice not stenosed, but valves incompetent from retroversion, and perforation of one valve. Pus in bronchial tubes. Large fatty liver. Spleen 11 oz.; minute (punctate) abscesses scattered over surface, and a few in its substance. No infarcts. Kidneys 12½ oz.; one large and pale, the other small, with dilated and injected pelvis. Bladder thick, urine foul. Brain: Embolism of right middle cerebral artery, with softening, which extended from cortex of insula to the internal capsule. Contents of the softened focus like sloughing brain matter, greenish yellow in colour, but not actual pus.

GROUP V.

CASE 47. *Ulcerative endocarditis (following endocarditis from chorea); mitral regurgitation; infarction of spleen and kidneys; acute basal meningitis; optic neuritis; tuberculosis of lungs; caseous mediastinal glands.*—Mary R—, æt. 15, was admitted under my care on June 26th, 1889, for pains in the limbs, pyrexia, and general malaise.

Family history.—Father has always been a healthy man. Mother has suffered from "consumption" as long as patient can remember. She used to attend the Brompton Hospital for diseases of the chest, and lately had been attending as an out-patient here. One of her brothers suffers from "boils" on the neck. None of her relations suffer from rheumatism.

Previous illnesses.—Patient had measles, whooping-cough, and scarlatina when a baby. Has never had rheumatism. Five years ago last August patient was in the hospital for St. Vitus's dance. She was in five weeks and got well.

History of present illness.—Four months ago she had an attack of "inflammation of the bowels." She was attended by a doctor, but has never been thoroughly well since. Three weeks ago she had an attack of diarrhœa. The week after this, June 13th, she had slight rigor, pain in her limbs, and retching without vomiting, and she complained of pain in her head. She went on with her work until June 18th, when she was obliged to go home on account of the pain in her head. She did not take to her bed until June 23rd, and on June 25th her mother got a doctor to see her, who advised her to come here.

Present condition.—The lips are rather dry, inclined to crack; fauces a little inflamed, causing some difficulty in swallowing; teeth regular; tongue pointed, bright red colour at the tip and sides; the dorsum is covered with a white creamy fur. There is no discharge from the ears.

Respirations 34 per minute; chest well formed, moves equally on both sides, tactile vocal fremitus equal on both sides. Air enters well in front. Some impaired resonance of the bases behind. No adventitious sounds.

Pulse 120 per minute, weak, compressible. Heart's impulse is felt in the fifth interspace in its normal position. The area of dulness is not increased; some slight tenderness is complained of on pressure over the area. There is a well-marked systolic bruit, heard best at the apex, and continued thence upwards into the axilla. This can also be heard at the base, but not so well. The second sound is normal.

The abdomen is not distended, and moves well. Liver dulness not increased; the spleen is tender on manipulation, and its dulness is a little increased anteriorly. Some tenderness on pressure in the right iliac fossa, but no gurgling.

Urine very acid, sp. gr. 1020; contains much albumen.

Joints not swollen or inflamed.

Ulcerative endocarditis was diagnosed, and she was ordered digitalis and quinine every four hours; milk diet, and four ounces of brandy.

June 28th.—Temperature this morning is 102·4°; pulse 120; resp. 46. Patient complains of constant pain in the head; she lies on the right side moaning, and often lifts her hand to her head. No paralysis. She was three times sick yesterday evening, and again this morning; bowels have not been opened. Photophobia present. The ophthalmoscope showed double optic neuritis. Temperature at 10 a.m. 103·8°; pulse 118; resp. 56. Patient was sick after taking some custard this morning. Pain still bad in the head. Dr. Washbourn examined patient's blood this afternoon for micro-organisms; three cultivations were taken.

30th.—Temperature at 10 a.m. 104·2°; pulse 116; resp. 66. Phenacetin, 8 gr., was ordered statim. Later on phenacetin, 10 gr., was given, and she was to be sponged with cold water when temperature reached 103°. The radial arteries feel of normal size, but the pulse in each is small, and can scarcely be counted. Pulse in each posterior tibial and in left dorsalis pedis. Nothing felt in right dorsalis pedis.

July 1st.—Temperature at 10.30 last night was 104·2°. Phenacetin, 10 gr., was given, and the temperature dropped to 103·6° at 11, 101·8° at 12.30. At 3.30 a.m. it was 104·2°.

She was sponged at 4 a.m. Temperature at 4.30 was 100·4°. This morning, 10.15 a.m., she lies in a collapsed condition on her back; surface cold, pulse almost imperceptible, face and lips livid, pupils dilated, eyes nearly closed, respirations noisy. The brandy was increased to 12 ounces. She died at 11.30 a.m.

Autopsy (by Dr. Perry):—*Brain.*—There was purulent basic meningitis, the greenish lymph and pus being found chiefly on the under surface of the pons Varolii and cerebellum. There was a flake of lymph and rather a large quantity of turbid serum in the lateral ventricles; the parts around, in particular the fornix, were very soft, and broke down under a gentle stream of water. No tumour and no cerebral abscess or infarct was found anywhere. The spine was not examined.

Pleuræ.—The right contained 6 ounces, and the left 7 ounces of serum. There were some adhesions on the right side.

Lungs.—Right normal, with no signs of compression. A caseous nodule at the back of the upper lobe, with grey miliary tubercles, and some pneumonic consolidation around it; and there were a few miliary tubercles at the upper and posterior portion of the lower lobe. The left lung was healthy, except for slight basic compression. Several caseous and calcareous mediastinal glands and the glands in the anterior mediastinum were large and succulent.

Heart.—10 oz. Muscle good, no infarcts. Fat small in amount. Coronary arteries good.

Attached to the auricular surface of the posterior flap of the mitral valve was a nodular vegetation about the size of a marble, firm, greyish in colour, and strongly adherent. On the anterior flap was a slightly thickened, rough patch, which seemed to be due to contact with the large vegetation. There was no perforation of the valve by ulceration, and its orifice was of normal size. Aortic valves normal. No vegetations.

A microscopic examination of the vegetation showed numerous aggregations of micrococci lying in round holes in the fibrin, as if they had liquefied it in their growth. The method employed to discover the cocci was to stain a fresh section with gentian violet, and then to decolourise by Gram's method.

Abdominal aorta normal, 1½ inches in circumference. There was a large plug of fibrin in the orifice of the cœliac axis, projecting into the lumen of the aorta. The projecting piece was the size of a pea. The embolus was just extending into the splenic, gastric, and hepatic arteries.

Liver 71 oz., large, but seemed normal.

Spleen 17 oz., large, and contained partly decolourised, firm infarcts.

Kidneys 11½ oz., contained a few small infarcts, which were pale and not purulent, though from one of them a small bead of what looked to be pus could be squeezed. I examined one of the infarcts microscopically, but could not find any micrococci, but this was not a softened or, to any degree, purulent infarct. There were no multiple punctiform ecchymoses in the kidneys. In fact, they looked quite normal organs, except for the infarcts. They were not scarred, and what infarcts there were had raised rather than depressed surfaces, in relation to the surrounding parenchyma.

Microscopical examination of the kidneys, stained with logwood, showed merely a badly staining infarct, with little or no small-celled infiltration round it. A vegetation stained in the same way showed only fibrinous material.

Case 48 (Dr. Goodhart, 1886–7).—Matilda S—, æt. 56. She had had a malignant tumour of the eyeball removed. October 19th, she had nodules in the skin of the calf, and one near the umbilicus. December, coughed up blood, and growth recurred in orbit. January, œdema of both legs; consolidation of base of left lung. Moderate pyrexia recorded during six weeks, September 30th to November 10th.

Autopsy.—Large masses of fungating endocarditis on aortic and mitral valves, and two small masses on tricuspid. New growth in left lung, surrounded by pneumonia. Nodules in liver and spleen. Left supra-renal converted into growth. The growth was found to be a myxomatous adenoma.

Case 49 (Dr. Hale White, 1889; see 'Path. Trans.,' vol. xli, 1890, p. 60).—George C—, æt. 25. Previous rheumatic fever. Admitted for headache, sickness, retraction of head, optic neuritis, with cerebral hæmorrhages, pain in cervical muscles, paralysis of left side of face and right side of tongue. Temperature between 100° and 101° throughout. Blood in the urine. Left side of tongue paralysed, and probably right side of the face. Death two weeks after admission.

Autopsy.—Hæmorrhage at base of brain, embedding the cranial nerves from the second to the twelfth. An aneurysm on each vertebral artery, with half an inch of the basilar, each compressing the hypoglossal nerve of its own side, and possibly the facial; that on the right side had ruptured. The subarachnoid hæmorrhage extended down the cord. Mitral orifice contracted, with recent vegetations containing micrococci. Infarcts in spleen and kidneys.

The aneurysms are preserved in Guy's Hospital Museum, No. 1519^{70}.

Case 50 (Mr. Davies-Colley, 1890).—James P—, æt. 62. Came in for abscess in Scarpa's triangle. Abscess opened; Oscillating temperature after this. After five days patient throwing himself about, clutching at clothes, groaning, delirious. Rapid breathing; passes urine under him; diarrhœa. Death on following day.

Autopsy. — Ulcerative endocarditis, with perforation of aortic valves; large heart, with excess of fat. Soft, œdematous lungs. Rather firm liver. Spleen large, but not unusually soft. Slightly granular kidneys. Disease of head of femur and acetabulum, apparently tuberculous, but no bacilli could be found.

Case 51 (Dr. Perry, 1888).—Maria J—, æt. 48. "Out of sorts" two or three days, then suddenly hemiplegia of left side; conjugate deviation of eyes to right. Loud mitral regurgitant murmur. Slow improvement; difficult feeding. A month later noisy at night, drowsy, semi-comatose, rapid pulse. Died six days later. Pyrexia began six days after admission, was moderate till six days before death, when it ranged between 101° M., and 104° E.

Autopsy.—Suppurative meningitis, more at base than vertex, and on vertex more on left than right side. No embolism found. Purulent fluid in lateral ventricles. Softening of right frontal lobe. Suppurative spinal meningitis whole length. Spinal bones and dura mater healthy. Œdema of lungs. Heart : Some soft vegetations on auricular surface of mitral, which was slightly thickened. No recent ulceration.

Case 52 (Mr. Lane, 1888).—Edward D—, æt. 19. A pail fell on his head and cut it. He came in two weeks afterwards with headache and optic neuritis. He was trephined.

Autopsy.—Heart 21 oz., hypertrophied and dilated ; aortic valves shrunken and incompetent, and a fibrous nodule on one cusp had rubbed an ulcer in its neighbour. Mitral orifice admitted two fingers, the margin beset with masses of vegetations ; aneurysmal pouching towards ventricular side. Tricuspid with a mass of vegetations and a friction ulcer. Pleural effusion and compression of bases of lungs. Three infarcts in spleen, one "of some date." Several infarcts in the kidneys. General suppurative pia-arachnitis ; no vessels blocked ; a small hæmorrhage on the surface of the left lenticular nucleus. No spinal meningitis.

Case 53. *Mitral endocarditis ; basal meningitis ; panophthalmitis ; infarcts in spleen and kidneys* (admitted to the Fulham Infirmary, under the care of Dr. Arnold Scott, who has kindly allowed me to publish the notes).—Henry F—, æt. 25. Good health till six weeks ago, when he had "congestion of the lungs," and though he got over this attack he had not been really strong or well since. He was up and about for a short time, but had to take to his bed again. On admission, March 25th, extremely pallid and much wasted, especially in the lower extremities. Full pulse ; a systolic bruit heard at the heart's apex, and one at the base. He has great pain in the neck, the muscles of which are rigid, and the head is retracted ; severe pain at the vertex. Conjunctiva of right eye much injected. Constipation. Basal meningitis was diagnosed.

March 28th.—Patient was constantly grinding his teeth. Twitching of face, chiefly on the left side. Muttering delirium.

29th.—Severe occipital headache. Still grinding his teeth;

unable to separate them more than half an inch. Tenderness on pressure of neck, especially over left sterno-mastoid, and twitching of left side of face, excited by firm pressure. Much wasting of limbs. No paralysis. Knee-jerks increased. Ankle-clonus present. Delirium in the afternoon. Chemosis of right eye, which is very tender to pressure. Vision defective. Urine, sp. gr. 1020, no albumen.

30th.—Increased chemosis and tenderness of right eye. Profuse sweating. Some delirium. Bowels confined, not answering to five grains of calomel.

31st.—Hiccough and incontinence of urine.

April 2nd.—Proptosis of right eye; no delirium.

3rd.—Profuse sweating. No delirium. No paralysis. Iritis, with pus filling pupil; emaciation and pallor extreme.

7th.—Typhoid condition; recurring delirium. Numerous petechiæ on trunk and limbs.

10th.—Died. The temperature did not show marked daily oscillations, but was high throughout, varying between 100° and 103°. The spleen dulness was increased, the mitral murmur increased in loudness, and ulcerative endocarditis was recognised.

Autopsy.—Vegetations on the mitral valve. Lower lobe of left lung airless. Infarcts in spleen and kidney. Brain: Basal meningitis involving pons and medulla oblongata. The vegetations were submitted to Mr. Targett, but he failed to find any micrococci.

The following is the case referred to on page 188.

Case 54.—*Aortic disease; pallor; high fever; delirium; ulceration of aorta and abscess of heart wall.*—James M—, æt. 27, was admitted on July 1st, 1890. He was a plasterer by occupation, and had been a heavy drinker. He contracted syphilis at the age of eighteen, had a sore throat, but no rash. He remained well till five years ago, when he had an attack of pleurisy, for which a blister was applied to the chest. He was ill a fortnight. In the beginning of June he got wet through one day. At night he had sharp pain in the left side, was short of breath, and panted all night. For three days he ate nothing, and he coughed up blood and mucus. He was in bed a fortnight, and went to work on June 23rd, but on the 28th he was ill again with "fresh cold," and took to his bed, and he has not been up since. He has not spat any more blood; but he coughs much, and brings up mucus.

On admission.—Well nourished. He lies on his right side to avoid pain in the chest. Face is pale, puffy, and much freckled; expression anxious; the lips and gums are pale; a yellowish tinge round the eyes. No œdema of the legs or other parts of the body. Pulse 100. Temp. 98·8°. Tongue furred, brownish yellow. Breath foul. Appetite very poor, but thirst complained of. Bowels loose. Splenic dulness not increased. Respirations 32, rather laboured. Coughed about every ten minutes, bringing up yellowish mucus. Voice husky and feeble. Sibilant and sonorous rhonchi all over chest, front and back. Behind, both sides are dull, right more than left; tactile vocal fremitus, and breath-sounds are deficient. Visible pulsation of carotids. Impulse of heart feeble; felt in the fifth space just external to nipple line. Cardiac dulness normal. Just a little after the systole a murmur of a booming or humming character occurs, and may be traced down to the apex. It is more booming over the sternum. In the aortic area there is a blowing systolic murmur traceable to the right clavicle. The pulse is full, feeble, but compressible, 100. There is no obstruction in either radial or posterior tibial arteries. Urine brown, clear, acid, sp. gr. 1030. No blood, sugar, or albumen.

July 4th.—Patient delirious all last night, quieter in the morning. Two motions last night and one this morning; they were liquid, of dark brown colour, like coffee, with particles of undigested milk. Face rather flushed. Antipyrin, gr. x. Ice-bag to head.

5th.—Was again delirious last night. He was sick twice. In the urine Ehrlich's test gives a bright ruby-red colour. Albumen is present. No rash. Normal pulse in posterior tibials. Motions watery, dark brown, with particles of undigested food. Ordered ammonia and senega, peptonised milk, antipyrin, gr. x statim.

6th.—Restless and delirious at night. Sick three times. Passed motions into the bed. Urine cloudy, dirty yellow, depositing reddish urates, acid, sp. gr. 1030, but no blood or sugar. Albumen 1·25 grammes per litre. A few pus-cells. Blood in the sputum.

7th.—Delirious, with noisy quick breathing. Not sick. Respirations 64 Strongly ammoniacal odour. Over the heart the systolic murmur is best heard at the base on the left side. Numerous râles all down right chest behind, with noisy, somewhat bronchial breathing at the mid-zone; on the left side loud sounds at apex, less loud at lower part, and at base deficient. Mist. Ammoniæ every two hours, antipyrin, gr. x statim, brandy, ℥iv. He remained in much the same state, delirious, but never quite comatose, and died at 11 p.m. The temperature throughout was high, varying between 101° and 104°. Several times it was above 104°, once only did it fall below 101°; an hour before death it was 106·2°.

Autopsy by Dr. Pitt.

Pleuræ.—A few scattered petechiæ and a few loose adhesions. The posterior parts of both lower lobes were tough, and the left was quite solid, sinking in water, and the right was partially so. This condition was due to the cardiac failure. Pus in the larger bronchi.

Heart 16 oz. There were about 2 oz. of clear serum in the pericardium, with some flakes of lymph; the surface of the aorta and the posterior surface of the auricles was rough with acute pericarditis. There were a few petechiæ. The right side of the heart was tough, and the cavity of the ventricle dilated. On opening the left ventricle the aortic valves were found to be thickened and shrunken, and incompetent. There were thick lines on the septum due to the regurgitant stream. The wall of the aorta just above the valves was thickened with atheroma for an inch immediately above the valves. Between the anterior and left posterior valves, immediately above them, was a small opening leading into a sac one inch in diameter, oval in shape, with a smooth wall, of a tough fibroid consistency, containing lymph, and no definite blood-clot. The sac lay in the tissue of the heart, and was not noticeable till the heart was opened. There was another sac in the wall of the left ventricle immediately below the other, which was filled with grumous contents, and which presented the appearance of an acute abscess, although it was rather an acute aneurism. The opening was into the aorta just above the right posterior valve, near the anterior. No cause for the ulceration of the aorta was made out, but there was calcareous material at the mouths of the sacs. There were no vegetations which were fungating, and no emboli. The condition was clinically that of ulcerative endocarditis, but not pathologically.

Spleen.—Small, firm, and free from infarcts. *Kidneys* normal and not tough. *Testes,* very slight fibroid change.

A CASE

OF

ILEO-COLIC IMPLANTATION,

FOLLOWED BY

RESECTION OF PORTIONS OF THE LARGE AND SMALL INTESTINE FOR THE CURE OF ARTIFICIAL ANUS, DUE TO GANGRENE AFTER STRANGULATED HERNIA.

By N. DAVIES-COLLEY.

The following case seems to me worthy of record, in the first place because the operation of ileo-colic implantation is a new one, and the conditions which call for it rare; in the second place because in one of the stages of the treatment my patient was left for a time with a fistula of a part of the small intestine which was cut off from the gastro-intestinal tract, and this afforded an opportunity to my colleague Mr. Tubby, and to Mr. Manning, to examine and analyse the secretion of the ileum. I have described the operations and subsequent treatment with some detail, and have added an explanatory diagram, in the hope that my account may be of some use to others who may have to perform a similar operation hereafter; for I found considerable difficulty in understanding the somewhat condensed descriptions which have been published in this country, of the various operations introduced by Professor Senn, of Milwaukee.

The account of the case is taken partly from my own notes, and partly from the careful reports of Messrs. W. C. C. Park, T. D. Manning, and A. J. Collis.

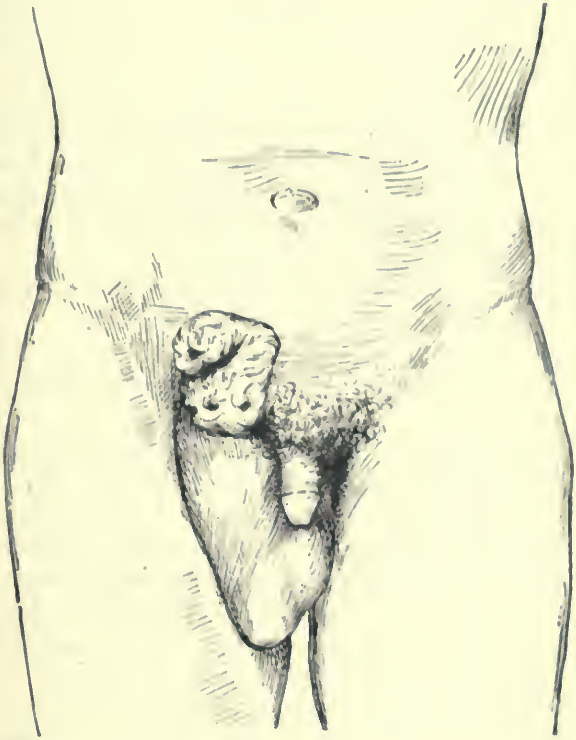
John L—, æt. 40, a labourer from Dartford, was admitted into Guy's Hospital under my care on May 27th, 1891, suffering from a large artificial anus in the right iliac region, through which all his motions passed. Eight months before admission he had been operated on for strangulated inguinal hernia, of two or three days' duration, in the West Kent General Hospital, Maidstone. The house surgeon there kindly sent us the following particulars.

"John L— was admitted some eight months ago with a strangulated hernia (inguinal). He has had a hernia for years, but never wore a truss; the hernia always used to go back at night. On admission he was very bad, vomiting, and vomit getting fæcal. Hernia was a little bigger than a man's fist. On exposing gut it was found to be gangrenous, so artificial anus had to be made. Sac was very thick. Bowel strangulated appeared to be cæcum, but I should not like to be sure, as appearance of gut was so altered by being gangrenous. A part of the circumference of the gut has sloughed away. The operation consisted of relieving strangulated gut and making artificial anus. The surgeon whom he was under was of opinion that it would not be any good trying to close up artificial anus, owing to loss of gut by sloughing, and nothing short of a resection would do him any good. Since operation he has suffered badly from prolapse of mucous membrane."

After leaving the hospital he wore an apparatus to collect the fæces, which all passed by the artificial anus. This apparatus occasionally caught the prolapsed bowel and gave him great pain.

On admission he was a strongly built and healthy-looking man. In the right inguinal region was a prominent elevation covered by smooth mucous membrane, and about three and a half inches in diameter. In this three openings could be detected; two were below, of which the inner only admitted the little finger a short distance, while the outer was considerably larger, and did not terminate in a *cul-de-sac*. These were evidently the orifice of the appendix vermiformis and the ileo-cæcal aperture. Above was a large protrusion of varying size, with a central opening. The prolapsed mucous membrane was therefore the lining of the cæcum and lower end of the ascending colon, while the protrusion was formed by the adjacent part of the ascending colon.

Fig. 1.



There was a frequent discharge of unformed motions through the ileo-cæcal opening, and none came away *per anum*. He had a large inguino-scrotal hernia on the same side, which could be readily reduced by taxis. The bowel, however, when reduced, appeared not to re-enter the abdomen, but to cause greater prominence of the prolapsed mucous membrane in the groin.

The size of the opening, the loss of part of the bowel wall, and the large associated hernia made it, in my opinion, impossible to close the artificial anus by the use of Dupuytren's enterotome, or any ordinary plastic operation. I decided, therefore, after consulting with some of my colleagues, to make such a communication between the ileum and ascending colon as would allow of the passage of the fæces, and at the same time to cut off from the intestinal tract the whole of the prolapsed bowel.

I was guided in my choice of an operation by the interesting paper of Mr. Jessett in the 'British Medical Journal' (vol. ii, p. 169, 1889). I also obtained from that gentleman some further useful information as to the exact mode of attaching the two portions of the intestine, and the subsequent treatment of the patient.

Two days before the operation a dose of castor oil was given, and the patient was put upon milk diet.

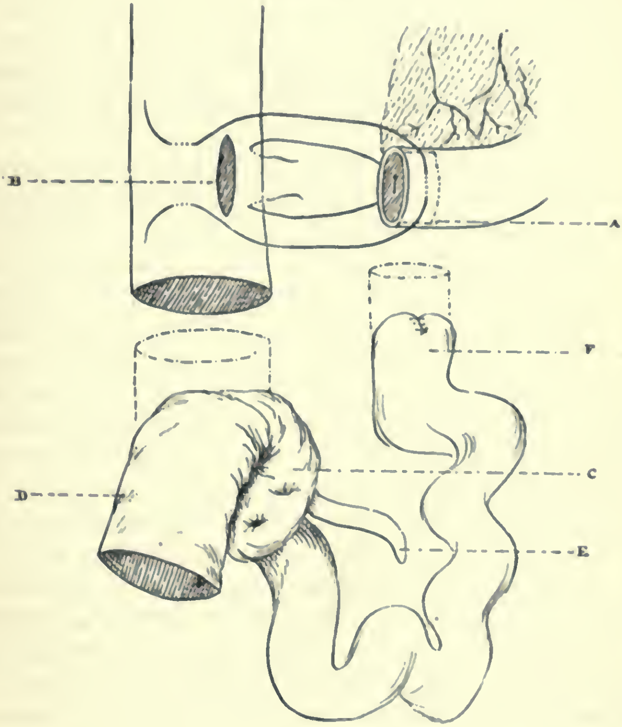
Operation (June 11th).—In the morning the parts of the bowel adjacent to the artificial anus were washed out with boracic lotion.

In the afternoon, the patient having been put under the influence of chloroform, which was followed by ether, the protrusion of mucous membrane was again carefully washed with soft soap, and afterwards with a weak solution of perchloride of mercury, and sponges were inserted into the ileum and ascending colon. A vertical incision about five inches long was made through the linea semilunaris down to an inch and a half above the protrusion.

Upon opening the peritoneum, the ileum came into view about three feet from the ileo-cæcal valve. I drew the lower part of the ileum out of the hernial pouch as far as I could, until I came to a point at which it was difficult to say which was bowel and which mesentery, the whole being obscured by bands of fibrous adhesions, and the mesentery being much thickened. Here I perforated the mesentery with the blunt point of a dressing forceps in two places, and encircled the ileum with two small pieces of drainage-tube, four inches apart. Each circle of tubing was drawn together with moderate firmness by a single knot, and then secured by the bite of a pressure forceps.

Having divided the intestine, and about an inch of the adjacent part of the mesentery halfway between the pieces of tubing, I cut a strip of india-rubber sheeting, five eighths of an inch broad by nearly three inches long, so that when united by sutures it would form a cylindrical ring (A, Fig. 2) exactly fitting the interior of the ileum. While my colleague Mr. C. J. Symonds, who kindly gave me the greatest assistance throughout the operation, was engaged in stitching this ring together, I proceeded to invaginate the distal portion of the ileum (F), and to secure its closure by a con-

FIG. 2.



tinuous gut suture passed through the serous and muscular coats. I then inserted the rubber ring within the proximal end of the ileum, and fastened it there by a continuous gut suture passed through its lower border and all the coats of the ileum.

Next I took two sutures of fine chromicised gut sixteen inches long, and armed with an ordinary straight sewing needle at each end, and passed them from within outwards through the rubber ring near its proximal border, and through the wall of the intestine which was in contact with it. The one pair of needles perforated the bowel about a quarter of an inch on either side of the attachment of the mesentery, the other pair upon the opposite surface at two points, half an inch apart.

The lower part of the ascending colon was now exposed. It was also bound down by adhesions, having bands containing large vessels crossing in front of it. I clamped it with two drainage-tubes, in the same way as the small intestine, but with an interval of five inches, and then divided the bowel between, about three inches from the distal clamp. A longitudinal cut (B), an inch and a quarter long, was next made on the inner side of the ascending colon close to the distal clamp. I now attached the ends of the sutures belonging to the proximal part of the ileum, those near the mesentery behind the cut in the colon and the other pair in front, passing each suture only through the serous and muscular coats of the colon about a quarter of an inch from the edge of the cut. Upon tightening the sutures the end of the ileum, stiffened by the short tube or ring of rubber in its interior, was drawn into the colon, invaginating the margins of the longitudinal incision, and each pair of sutures was then knotted, but not so tightly as to cause puckering up of the half-inch of bowel wall which each embraced. With my finger introduced through the transverse section of the colon I could feel the entering piece of the ileum, and facilitate its introduction and adjustment. As, however, gaps were left both above and below the junction, I had to complete the adjustment by five or six Lembert sutures in each of these situations.

I now proceeded to invaginate the open end of this portion of the colon, and to stitch it up with interrupted Lembert sutures. This proved very difficult to complete in a satisfactory manner, because the coats were stiffened by old inflammation, and the tissues so altered in appearance that it was sometimes doubtful whether I was dealing with intestine or mesentery.

The proximal end of the colon had next to be attended to. Having removed the tubing with which it had been clamped, and the sponge which had been placed in the opening at the beginning of the operation, I passed my finger in by the artificial anus, and drew down this part of the bowel (D), turning it inside out. Then after stitching it up by a continuous gut suture passed through all the coats of the intestine, so as to bring the serous surfaces, which were now inside, into close contact, I cut it away a little external to the suture, removing about two inches of ascending colon. Finally, I took away the remaining clamps of tubing and the sponges with which the edges of the abdominal wound had been guarded, and examined carefully to see if there were any leaking of fæcal matter. None was found; so after sponging the adjacent peritoneal surfaces the wound in the parietes was closed with seven strong gut sutures, and a dressing of carbolic and sal-alembroth gauze was applied. The prolapsed bowel of the artificial anus (C) was dusted over with iodoform and dressed with sal-alembroth gauze.

The operation took more than two hours. The chief reason for the long time consumed was the great number of sutures which had to be inserted. The invagination of the distal end of the ascending colon was especially tedious, for when it was almost complete small pieces of mucous membrane seemed to protrude afresh, and to require the application of more Lembert sutures. It would have been better to have allowed a longer piece of colon than three inches for the ileo-colic opening and the new cæcum. At the end of the operation the patient was rather cold, but his pulse, though small, was regular and not above 100. He was ordered Tinct. Opii ℳxv bis die, and an enema containing four ounces of peptonised milk, with some Brand's essence and half an ounce of brandy every four hours.

12th.—Has had a good night, having slept about five hours without waking. Complains a little of flatus. Abdomen not swollen. To-day he was given peptonised milk and a little Brand's essence, a teaspoonful at a time, by the mouth. Altogether he had by the mouth two and a half ounces of milk and two drachms of Brand's essence. He passed sixteen ounces of urine.

13th.—Comfortable, but complains of rumbling in the bowel; no flatus, however, has escaped *per anum*. He was allowed more peptonised milk by the mouth, viz. two drachms every half-hour. He took altogether six ounces of milk and six drachms of Brand's essence. As he was thirsty he was given an enema of fifteen ounces of warm water in addition to the nutrient enemata. The dressing upon the artificial anus was changed.

14th.—Passed a good deal of flatus *per anum* for the first time since the operation. The tincture of opium had been stopped.

16th.—Some pain about 7 a.m. in right iliac region. He passed some small loose motions of natural appearance to-day. The enemata were stopped yesterday. He has taken more than two pints of milk by the mouth in the last twenty-four hours. He passes from ten to fifteen ounces of urine daily.

17th.—Operation wound dressed for the first time. There is a little bloody serum in the dressing, and no smell. Three sutures removed. The dressings on the artificial anus were somewhat foul. Abdomen generally neither swollen nor tender. Temperature, which has hitherto remained low, went up to 101·6°. Pulse, which has been good, and about 80 ever since the operation, is now 88.

18th.—Temperature down to 99°. A good deal of pain in the right iliac fossa, which is relieved by the passage of flatus. He takes more than two pints of milk daily, with the yolks of two eggs and barley water.

20th.—Second dressing of the wound. Good apposition. All the sutures removed. Discharge of some brownish serum by one of the suture holes.

21st.—Temperature again rose to about 100°, and in the night a good deal of foul discharge came through the dressings.

22nd.—Has been easier since the discharge came away. Wound dressed, and bad-smelling fluid very like thin fæcal matter was found to be oozing from the middle of the wound. General condition good.

23rd.—Passes somewhat pale pultaceous motions *per anum.* From the wound, which is now dressed with boric lint, turbid fluid without any air can be pressed.

29th.—Bowels not moved from the 26th till to-day, when he passed a large pale solid motion, in which was contained the ring of rubber sheeting still fastened together by the gut sutures. There is now hardly any discharge from the abdominal wound.

July 6th.—Allowed to sit up a little.

29th.—A second operation was performed under ether, with the object of removing the prolapsed bowel with that part of the ileum which by the previous operation had been disconnected from the rest of the intestine. Having divided the skin close to the margin of the artificial anus all round, I opened the peritoneal cavity and proceeded to dissect through the mesenteric attachments of the bowel. These were very thick and vascular, there being numerous fibrous adhesions as well as the normal structures of the mesocolon and mesentery. More than twenty small vessels had to be tied. The piece of ileum was eight or ten inches in length, and I found that its proximal end was in close connection with the back of the artificial ileo-colic opening made in the preceding operation. I dared not pull this portion down for fear of tearing an opening into the pervious part of the bowel, so leaving this I proceeded to clear away as completely as I could the thick peritoneum which lined the hernial sac in the scrotum. Finally, I brought together the edges of the lower part of the opening with three deep button sutures, and I brought out the piece of the ileum which I had not ventured to remove through the upper portion of the wound, and having cut it through I attached the edges of its mucous membrane to the skin. It formed a *cul-de-sac* nearly three inches long. The operation was performed without the spray, but with careful antiseptic precautions. It lasted two hours. I hardly saw any other part of the bowel than that which I removed. The portion removed consisted of ileum, cæcum, with its appendix (E), and a small portion of the ascending colon.

The temperature rose above 103° the next day, and three days after the operation, at the first dressing, there was a good deal of foul discharge, suggestive of an escape of fæcal matter. This continued for a few days, and air was seen to escape with the discharge, so it is probable that there was some small communication with the interior of the pervious bowel. In eight days, however, from the operation the discharge lost its fæcal character, and the wound rapidly healed, with the exception of the fistular opening at its upper extremity, which communicated with the separated portion of the ileum. From this time there was a constant discharge of a watery fluid, the examination of which by Mr. Tubby and Mr. Manning is described in the annexed paper. He was soon able to get about, but the weakness of the abdominal wall left by the wound and by the artificial anus required the support of a belt, while a tendency to the return of the scrotal hernia had to be kept in check by a truss.

The continued flow of fluid from the intestinal fistula, and the prolapse of the mucous membrane, made the patient so uncomfortable that on December 9th a third operation was undertaken. At this time the fistula consisted of a canal about an inch in diameter by three and a half inches in length. Under ether the mucous membrane was separated from the skin at the orifice of the fistula, and it was then carefully dissected from the interior of the *cul-de-sac.* I thus removed the whole of the mucous membrane, which came away like the finger of a glove, leaving the other coats *in situ.* The only point at which there was any difficulty was at the extremity of the *cul-de-sac,* where I had to tear through some adhesions. The hæmorrhage was rather free from the part that was near the junction with the skin, as I had carried my dissection so deeply as to wound some vessels in the muscular coat, but as long as I kept in the submucous tissue and used blunt instruments I got hardly any hæmorrhage. In the evening the wound began to bleed again, and the house surgeon, Mr. Rygate, was obliged to plug the opening before he could arrest the hæmorrhage. This caused some delay in healing, but in a month's time the wound was practically well. There was some difficulty in fitting an efficient truss and belt, but this was finally accomplished, and the patient went out well in March, 1892.

In none of the operations was the carbolic spray used, but great care was taken to render and keep the parts aseptic. Fig. 2 represents diagrammatically part of the procedure in the first operation. The bowel indicated in the lower part of the figure was first separated from the intestinal tract, and finally removed in the second and third operations, with the exception of the muscular and serous coats of rather more than three inches of ileum.

A RESEARCH

ON THE

PROPERTIES OF PURE HUMAN SUCCUS ENTERICUS,

WITH A FEW NOTES ON

ABSORPTION BY THE BOWEL AND INTRA-ABDOMINAL PRESSURE.

By A. H. TUBBY, M.S., and T. D. MANNING

(*From the Physiological Laboratory*).

In writing an account of our work we are anxious to express our gratitude to Mr. Davies-Colley, from whom for over a period of three months we received help in every way, and were by him afforded unusual facilities of access to the patient in making daily observations; without this kindly assistance the work could not have been undertaken. To Mr. F. Gowland Hopkins, Gull Research Scholar, Guy's Hospital, our thanks are due for much information in matters of chemical technique, and for the practical assistance he gave us in the occasional use of a portion of Dr. Debus's laboratory apparatus.

An account of the case from which we obtained the succus entericus is written by Mr. Davies-Colley, and precedes this article in the present volume of the 'Reports.' On reading the history of the patient and the operations performed on him, it is evident that absolutely *pure* succus entericus flowed from a fistula communicating with a portion of the ileum about three and a half inches in length, entirely shut off from the remainder of the short-circuited small intestine. The isolated part of the bowel was situated about eight inches from the ileo-cæcal valve. Hence there was no admixture of bile, pancreatic juice, or the altered products of digestion in the succus entericus obtained. It might be urged that the mucous surface of the severed intestine lacked its natural stimulus in the passage and absorption of partially digested food, and that the secretion was not trustworthy. To us it seemed that the constancy of what we regard as the natural reactions and qualities of the fluid even to the fourth month is a sufficient reply to any objection on this score. The blood-supply to this particular part of the gut was good, and the mouth of the fistula maintained a fairly constant appearance from first to last, merely becoming congested either from slight prolapse or manipulation. The health of the patient was good, except during the third month, when some anasarca developed; but the appetite was well maintained, and there was no loss of weight. A point of some importance requires emphasis, viz. that there was no partial digestion or ulceration of the skin around the orifice, thus completely contrasting with the discomfort and the soreness of the parts around a gastric fistula.

Our observations extended from August 28th, 1891, to December 9th of the same year, and the secretion was regularly collected and examined by us for 104 days. On some days two collections were made. The method of collecting the juice was generally by means of a sponge. Our hands underwent a thorough cleansing and sterilisation previously in a $\frac{1}{2000}$ solution of perchloride of mercury before removing the sponge. The skin around the fistula was washed daily with warm boracic lotion. Two sponges were kept at work, relieving each other, and were rendered aseptic by being washed in a $\frac{1}{1000}$ solution of corrosive sublimate, then soaked in sterilised water until the washings ceased to give a reaction for mercury salts, and finally placed in an incubator until required for use. With the object of avoiding fallacies, the last washing of the sponges was tested for proteids sugars starches sulphates phosphates and chlorides, and gave negative results. The fistula and adjacent skin having been carefully cleansed, a sponge was placed on it, then some clean gutta-percha tissue, a pad of perchloride wood-wool, and the whole fixed by a bandage. As a rule the sponge was changed once in twenty-four hours, but on about twenty-five occasions twice in that time. We found it at the end of the above-mentioned times *free* from odour of decomposition, soaked with intestinal juice, and partially covered with thick tenacious mucus. Other methods of collection were tried on various occasions instead of the ordinary sponge; asbestos wool previously heated in a Bunsen flame was employed, but the fluid stank so much that we were compelled to cease from using it. A specially designed bottle of small size, combined with a truss, was also fitted and used, but was, at first, not entirely satisfactory, as hæmorrhage from the everted mucous membrane sometimes took place. We then had some more bottles made with larger mouths, and they proved satisfactory in every way. They were hemispheroidal in shape, rather flattened on the outer surface, and with a large round opening on the inner surface, surrounded by a thick projecting lip. One of these was fixed by a simple arrangement into a broad elastic belt, which the patient wore without discomfort from 11 a.m. to 6 p.m.; in fact, at one time, for a period of ten days he was employed on some light manual labour. The bottle was replaced by a sponge at night.

There was no difference in the appearance of the secretion collected by these methods, except that there was perhaps more mucus in that collected by the bottles. Unfortunately there was great difficulty in procuring the latter, so that they were not used so often as we wished.

The fluid was obtained from the sponge by squeezing it as soon as possible into a clean evaporating dish—the mucus having been first picked off, and either kept for separate use or added to the secretion. It was then poured into a test-tube previously washed out with absolute alcohol, a stopper of charred cotton wool was inserted, and the tube placed in an incubator at 37° until required.

Literature.

A *résumé* of work done on this subject up to 1882 is given by Dana,[1] and we take the liberty of quoting his abstract. Busch examined the juice from an intestinal fistula high up in the intestine of a woman. Proteid bodies were dissolved with the appearance of putrescence and the odour of ammonia; starch was acted upon more energetically than proteid bodies; fats were unaffected, and not absorbed unless mixed with bile; cane-sugar was unaffected. Ewald[2] found that the succus entericus, taken from a fistula in man, turned albumens to peptones, starch to sugar, and formed an emulsion from fats. Demant[3] obtained the juice from a fistula low down in man: on proteids no action occurred; starch was changed to grape-sugar; fats with fatty acids were emulsified; neutral fats were not affected, and cane-sugar was changed into grape-sugar. Thiry obtained the juice from an artificial fistula in animals. He found that fibrin crumbled in the normal secretion, but this did not occur if the juice were neutralised or modified; coagulated white of egg and red muscle were unaffected; starch was not converted into grape-sugar; fats were unaffected; no change in cane-sugar took place after some hours of digestion; and on grape-sugar no fermentative influence was observed. Leube found crumbling of fibrin, no change in starch, conversion of cane into grape sugar, and formation of lactic and butyric acid from grape-sugar as the result of fermentative action of vibriones. Wittich found an energetic diastatic action on starch in one instance. Paschutin found that starch and cane-sugar were converted into reducing sugar, and fats were imperfectly emulsified. Bernard thought that the chief action of the secretion of the small intestine was conversion of cane into grape sugar.

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[1] 'Philadelphia Med. News,' 1882, vol. xli, p. 59.

[2] "Ueber des Verhalten des Fistelsekret, &c.," 'Virchow's Archiv,' lxxv, 3, p. 409, 1879.

[3] "Ueber die Wirkungen des menschlichen Darmsafts," 'Virchow's Archiv, lxxv, 3, p. 490.

Garland and Masloff[1] found that albuminoid substance was attacked, and starch turned into grape-sugar, Masloff also noting that peptones were produced from fibrin. Dana, by injecting albumen solutions into the intestines of dogs and horses, found peptones produced, also that starch was changed into grape-sugar. Quincke, by using glycerine extracts of mucous membrane, found that fibrin was dissolved, but not converted into peptones, and starch converted into reducing sugar, and fats unaffected. Schiff noted that proteids were merely dissolved, and he thought that the action on starch was nearly as energetic as that of pancreatic juice, while oils were emulsified. Brown and Heron[2] found that starch was converted into grape-sugar, and maltose also into grape-sugar. They further elucidated a most important fact, viz. that while the pancreatic diastase turns 80 per cent. of starch into maltose, the intestinal juice turns both into dextrose. Our experiments support these results. Subsequently to 1882, Solera[3] isolated a piece of the gut by ligatures, and killed the animal six hours later and made trials of the absorptive powers of the gut. Hermann, in the 'Archiv für Physiologie,'[4] gives an account of sundry experiments conducted by him on dogs. Röhman[5] has given a *résumé* of experimental work on the succus entericus of animals.

General Remarks on the Qualities of the Succus Entericus obtained by us.

Amount.—In an earlier stage of collection the average quantity yielded by the small portion of the bowel under observation was 27 c.c., but in the late stages, during the last month, 24·5 c.c. The largest amount collected in one day, in two instalments, was 46 c.c. When we could be sure that none had been lost by misadventure, we considered 35 c.c. a good, and 19 c.c. a bad yield, and this is probably too small for the normal amount, since there was an entire absence of stimulus to any part of the mucous membrane, except the half to three quarters of an inch which protruded through the fistula. The causes of the variation we were unable to ascertain. Apparently feeding had nothing immediately to do with it, as the man had his meals with hospital punctuality, and we were unable to discover the receipt by him of extraneous or surreptitious supplies.

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[1] 'Zum Dünndarmverdauung Untersuch aus dem Phys. Instit. Heidelberg,' ii, 3, p. 290, 1878.

[2] 'Proc. Roy. Soc. Lond.,' xxx, 393.

[3] 'Archives Italiennes de Biologie,' 1884, vol. v, p. 20.

[4] Ibid., vol. xlvi, p. 93.

[5] Ibid., vol. xli, p. 411, *et seq.*

The *specific gravity* was taken of the first thirty collections by actually comparing its weight with that of an equal quantity of distilled water; the average was found to be 1006·9 (water 1000), the limits being 1001·6 and 1016·2. On two occasions the specific gravity was taken of the secretion collected by means of the bottle, and it was then 1003·2 and 1010·3.

Appearance of fluid.—It was generally opalescent, often having, however, a brownish shade, not acquired from the sponge. Occasionally it became tinged with blood, due to some oozing from the surface of the everted mucous membrane; and caused by friction and manipulation. On testing this blood-stained fluid no peptone was found in it. The significance of this point will be later insisted on.

Odour of fluid.—This was constant and characteristic, a peculiar faint alkaline smell, rendered more marked by the addition of strong sulphuric acid, and not unlike that of blood.

On keeping the fluid at 37° C., a few of the specimens became decomposed in two to three hours, but the greater majority remained quite sweet for twenty-four, and we have kept some in cotton-stoppered tubes for several days. Sometimes a few drops of alcoholic solution of thymol were added, but we had reason to think that the alcohol interfered with ferment action, so we trusted to our antiseptic precautions during collection.

Reaction and chemical composition.—It was invariably alkaline. We made an effort to estimate the alkalinity in terms of sodium carbonate and acetic acid, but our results differ widely, and we hesitate to quote them. The alkalinity was probably due to a considerable quantity of carbonic acid in combination with alkaline metals. We found that effervescence occurred on adding strong HCl, and that the gas given off gave the ordinary reactions of CO_2. The fluid gave the biuret and xanthoproteic tests. With Millon's fluid, acetic acid, and ferrocyanide of potash the usual proteid tests were obtained, and Adamkiewicz's test was specially well marked. It failed to reduce Fehling's solution, nor did it alter the colour of iodine solution. It deepened a solution of perchloride of iron, and gave Uffelmann's test (carbolo-chloride) for lactic acid immediately on collection and twelve hours later. We were unable to undertake the estimation of whatever lactates may have been present by employing the more reliable method of crystallising out. An unfailing characteristic of the fluid was the persistent presence of a large quantity of mucin. The *rôle* this plays we made an effort to determine, and we noticed that after twelve hours' keeping the fluid Uffelmann's test was more pronounced the greater the quantity of mucin present.

Microscopically.—The fluid showed a few leucocytes, some nucleated columnar cells immediately after collection, and no bacteria in any of the eleven specimens examined.

CONVERTING POWERS OF HUMAN SUCCUS ENTERICUS.

Summary.

On proteids.—No change was produced on *serum-* or *egg-albumen, fibrin* or *casein.* The gelatinisation of boiled solutions of *gelatin* was prevented, but no gelatin peptone was found.

On fats.—These were found to be emulsified and saponified.

On carbo-hydrates.—Cellulose was totally unaltered, even after prolonged periods. *Cane-sugar* was converted into *grape-sugar,* not merely into maltose, in more than 60 per cent. of the experiments. The greatest amount of grape-sugar formed did not exceed on any one occasion half the cane-sugar employed. *Starch* was converted into *grape-sugar* in 56·5 per cent. of the experiments, but the proportion of starch so converted was greater than the proportion of cane-sugar. *Erythrodextrin* appeared during the conversion.

Maltose was converted into *grape-sugar.* This we proved by employing the phenyl-hydrazin test, and by estimating the comparative reducing powers of standard solutions of maltose in water, and maltose acted on by succus entericus. A very large amount, if not all of the maltose, is thus altered, so that it may be affirmed that a very important function of the succus entericus is to complete the salivary and pancreatic digestions.

On milk.—Casein is precipitated, and the milk becomes acid.

Ferments.—We obtained a glycerine extract from the mucous membrane which converted cane-sugar, starch, and maltose into glucose, and prevented the gelatinisation of boiled solutions of gelatin.

Detailed Account of Experiments.

On proteids.—In our earlier experiments the results were vitiated by faulty dialysers, but this was remedied by using cartridge-paper dialysers sausage-skin shaped, taking care that there was no leakage of any kind, and bearing in mind that they may allow albumen to pass through them after twelve to twenty-four hours. We were also confronted by another difficulty in the discovery that if ammonium sulphate be used to separate peptones from other proteids, the ordinary biuret reaction is not reliable, even though a great excess of caustic potash be added, as the pink colour typical of peptones is most capricious in its appearance. We were also heedful of the possibility of an albumose or peptone reaction being obtained, owing to the action of putrefactive bacteria on albumens if the experiment were prolonged unduly. In connection with any possible proteolytic powers of the intestinal juice, we insist upon the absence of partial digestion of the mucous membrane or skin throughout the period of observation.

Experiments with blood-serum.—On eleven occasions the mucous surface bled owing to various circumstances, such as digital examination and congestion. On careful dialysation and re-dialysation no peptone was found at any time. We made two direct experiments in test-tubes with a solution of serum-albumen, and obtained no evidence of conversion. Alcohol failed to give a precipitate with the doubly dialysed fluid, nor was any biuret reaction obtainable.

Experiments with egg-albumen.—Six trials were made in test-tubes, and again no change occurred. There was no attempt at solution of the pieces of coagulated albumen.

Experiments on fibrin.—At first we tried to estimate any change in fibrin by staining it with carmine, and noting if the succus entericus became red after some hours. This we found involved a fallacy, since alkalies or mixtures of alkalies and fibrin dissolved out the carmine, probably from the solvent action of alkalies *per se* on fibrin.

Experiments on casein.—Freshly prepared casein and Grubler's "casein purissimus" were mixed with succus entericus in test-tubes, and kept at 37° for twenty-four to forty-eight hours, but no conversion results were obtainable.

Experiments on alkali-albumen.—The albuminate was prepared fresh by acting on serum-albumen with a weak solution of caustic potash at 37° C. for eight hours, and then submitting it to the action of the juice. On three occasions no peptones were found, and in twenty-four hours' time the mixture stank.

Experiments on some of the Albuminoids.

1. *On gelatin.*—Various trials were made, and from the results two conclusions were drawn: (*a*) that succus entericus prevented the gelatinisation of gelatin; (*b*) that any change of gelatin into the so-called gelatin-peptone was open to doubt owing to some decomposition occurring during dialysation. With reference to the first point, an account of the experiments is subjoined.

Experiment 1 (September 12th).—Fresh succus entericus was added to a solution of gelatin in a cotton-stoppered, clean test-tube. In a few hours it was observed that the gelatin was broken up in some way, and formed with the juice a turbid liquid.

Exp. 2 (September 27th).—A solution of gelatin was prepared by boiling, and cooled succus entericus was added to one half (*a*), and an equal amount of water was added to the other half (*b*). Both were placed in the incubator for seventy-two hours, and then transferred to the ice chest; then (*a*) failed to solidify, whilst (*b*) formed a good jelly.

Exp. 3 (September 29th).—A similar experiment, but the test-tubes were taken out of the incubator at the end of four hours. The result was the same.

Exp. 4 (October 2nd).—Similar in every way to the last.

Exp. 5 (October 14th).—On the same lines as the three preceding, but kept in the incubator for forty-eight hours. Result, the same.

Exp. 6 (October 20th).—Similar to the preceding, but kept in the incubator for twenty hours. Result, the same.

Exp. 7 (October 20th).—The solution of gelatin was not heated before use, but after eighteen hours' action the result was the same.

Exp. 8 (October 21st).—Similar to Nos. 2—6, but the succus entericus was allowed to act for only one hour. The mixture was then found to gelatinise as well on cooling as did that in which there was nothing but isinglass and water.

Exp. 9 (October 25th).—On this occasion the boiled gelatin solution was divided into four portions. To one portion water was added, to another weak acetic acid, to the third sodium carbonate (·05 per cent. solution), and to the fourth some succus entericus two hours after collection. At the end of twenty-four hours none of these would gelatinise on cooling.

Exp. 10 (December 2nd).—On this occasion the gelatin solution was divided into three parts, and the substances added were water, sodium carbonate (1 per cent. solution), and succus entericus, all in equal amounts. After half an hour's action in the cold the contents of the test-tube which contained succus entericus were found to be liquid, whilst the other two had solidified. All three were then placed in the incubator for twenty-four hours, and then transferred to the ice chest, when it was found that the first two solidified whilst the third did not.

With reference to the second point, that is the formation of gelatin-peptones, some of the products in the above ten experiments were tested for peptone with the following results, dialysation being used in each case :

Exp. 5 (48 hours).—Good peptone reaction, but after twenty-four hours in the dialyser the liquid was found to be decomposing.

Exp. 6 (20 hours).—No peptones were found. In this case thymol was added when the products were placed in the dialyser.

Exp. 7 (18 hours).—Good peptone reaction. In this case, it will be remembered, unboiled gelatin was used, and the presence of bacteria probably caused some decomposition with formation of peptones.

Exp. 9 (24 hours).—No peptones were found.

In four early experiments, in which boiled gelatin was acted on by the secretion for periods of twenty-four, forty-eight, seventy-two, and ninety-six hours, no peptones were found, but we were then using the method of precipitating everything but peptones by ammonium sulphate; and though we did not in any case obtain any peptone biuret reaction, it is possible that peptones may have been present, and that the test failed owing to the presence of the large amount of ammonium sulphate, as was certainly the case in testing for ordinary peptones. We do not, therefore, place much reliance on these four experiments, but in the other four cases quoted above (Nos. 5, 6, 7, and 9) it is clear that no peptones were found when there was no decomposition.

Experiments on Fats.

The question we set ourselves to answer was whether the succus entericus was capable of emulsifying and saponifying fat, and we came to a positive conclusion.

Some considerable difficulty was encountered in obtaining a perfectly neutral fat, it being evident that a fat containing free fatty acid could be a great cause of fallacy. We employed a perfectly neutral specimen of cod-liver oil from Messrs. Allen and Hanbury.

The method of estimating the soaps formed consisted in allowing the succus entericus to act on a neutral oil for a given period at a temperature of 37° C. in clean cotton-stoppered test-tubes, then to place the mixture in a dialyser for seventeen hours, and to test the dialysate in two ways, either by what we venture to designate the "acid and ether" method, or by the addition of a few drops of osmic acid. The acid and ether process consisted in boiling the dialysate with strong hydrochloric acid for some time, cooling, shaking up with ether, pipetting off the ether, and noting whether any fat-like residue was left on a watch-glass, or a stain formed on blotting-paper. A simple and very reliable method consisted in adding a few drops of a 1 per cent. solution of osmic acid to the dialysate, noting any ensuing blackening, not merely darkening of the mixture. A solution of osmic acid was found by check experiment to blacken, not merely darken from exposure to daylight, a solution of pure soap; and we are of opinion that, in the case of fat, the osmic acid affects not the glyceryl radicle, but the fatty acid in combination.

Exp. 1 (November 13).—To 3 c.c. of succus, twelve hours old, but kept in clean cotton-stoppered test-tube at 37°, was added 1 c.c. of neutral oil, and the mixture kept at 37° for thirty minutes. The mixture was placed in a dialyser, and the dialysate tested by the "acid and ether" method gave a fatty deposit on a watch-glass.

Exp. 2 (November 11th).—3 c.c. of succus entericus (twelve hours old) was added to 1 c.c. of neutral oil. At a temperature of 37° C. the mixture gave in three minutes a slight acid reaction, in six minutes more pronounced reaction, and in thirty minutes a good red colour on litmus paper. The mixture was put to dialyse, and gave acid and ether test. Also 1 c.c. of the dialysate turned jet-black in twenty-four hours after the addition of 3 minims of a 1 per cent. solution of osmic acid. On using 2 c.c. of oil with the same quantity of succus entericus of same date, equally good evidences of acid reaction were obtained on litmus paper.

Check experiment.—3 c.c. of ·05 per cent. solution of sodium carbonate added to 1 c.c. and 2 c.c. of neutral oil; blue litmus paper was reddened, but not vividly or so rapidly. Change of colour did not in this case occur until twelve minutes had elapsed (in the case of succus entericus reddening in three minutes). The sodium carbonate was allowed to act on oil for the same length of time and under the same circumstances as in the succus experiment. On testing with "acid and ether" process, less signs of soap having been formed were obtained, and the dialysate gave *no* blackening with osmic acid solution.

Exp. 3.—3 c.c. of succus, eight hours after collection but carefully preserved from decomposition, were added to 2 c.c. of neutral oil, and the mixture kept at 37° C. for thirty minutes, and then dialysed for eight hours. The dialysate gave the soap test with "acid and ether" process. Also the addition of osmic solution turned 1 c.c. of the dialysate brown in six hours. In twenty-four hours the colour deepened to jet-black.

Check experiment.—Doubtful soap reaction with 3 c.c. of sodium carbonate solution, ·05 per cent., and *no* darkening or blackening with osmic acid.

Exp. 4.—Succus entericus 3 c.c. of 28th November added to 2 c.c. of neutralised oil, and kept at 37° for thirty minutes, then dialysed for twelve hours, gave both "acid and ether" and osmic acid tests for soaps.

Check experiment.—Sodium carbonate solution ·1 per cent. and ·25 per cent. added to neutral oil, and kept at 37° for thirty minutes, gave doubtful evidence of formation of soap with acid and ether method, but the dialysate was unaffected by the addition of osmic acid.

These experiments show that succus entericus contains a something which splits fats up and saponifies them. That something is more rapid and powerful in its action than ·05, ·1, and ·25 per cent. solution of sodium carbonate. Whether it be a ferment which gives rise to this change in fats we are not prepared to say, since we have failed to isolate it.

A further point might be noticed. It was found that the emulsion formed by adding succus entericus to oil was finer and more durable than any emulsion obtained by adding ·05, ·1, and ·25 per cent. solutions of sodium carbonate to equal quantities of neutral oil.

Experiments on Carbo-hydrates.

On cellulose.—The form we used was cotton-wool, well purified and washed in distilled water. A small quantity of this was placed in distilled water for twenty-four hours, and then Fehling's and Parry's tests for reducing sugar were tried. No reduction of copper occurred.

Exp. 1.—Succus entericus allowed to act on cotton-wool for twenty-four hours at 35° C. On testing, no reducing sugar was found, and the microscope showed no changes in the outlines of the fibres.

Exp. 2.—A repetition of Experiment 1: examination on the sixth, eighth, and twelfth days showed no change microscopically or with Fehling's solution.

Exp. 3 (September 3rd).—Absorbent cotton-wool placed in succus entericus for seventy-two hours. We thought that the cotton-wool fibres had undergone a slight change in appearance when seen under the microscope. There was, however, no sign of erosion, nor were the fibres swollen, but merely slightly granular. This might have been due to the prolonged soaking, as a similar appearance was observed after eight days. But even after so prolonged a period no reduction of copper from Fehling's solution could be obtained.

On cane-sugar.—It has been generally accepted that succus entericus has an inverting action on this substance, and we subjoin a table of our results.

Table I.—*Table of Results of Action of Succus Entericus upon Cane-sugar at 37° C.*

Substance acted on.	Form of secretion.	Number of experiments in which reduction obtained.			Total number in which a reduction was obtained	Number of failures.	Total number of experiments.	Remarks.
		In 12 hours and under.	In 12 hours to 24 hours.	Over 24 hours.				
Cane-sugar	Succus entericus	10	19	7	36	24	60	
"	Succus entericus without mucus	0	2	0	2	4	6	
"	Mucus only	4	5	2	11	4	15	Of failures, two were when water in which mucus had been washed was used.
Totals .	.	14	26	9	49	32	81	

The experiments were carried out in clean cotton-stoppered test-tubes at 37° C.

The cane-sugar was dissolved in cold distilled water and then boiled, so as to form a standard solution of 1 gramme of sugar to 10 c.c. of water. With this solution we made many experiments. We found that the mere boiling, if prolonged, of a solution of cane-sugar and Fehling's liquid was sufficient to give a slight reduction. If, too, cane-sugar were boiled in an acidulated or alkalinised water, the suboxide of copper appeared more readily and in greater quantity. Bearing this in mind, we have treated all cases in which the reduction did not occur *at once* on heating as negative experiments.

It is evident from this table that we obtained an inverting action on cane-sugar forty-nine times out of eighty-one experiments, or 60·5 per cent. of successes. The quickest time of inversion was three minutes; on several occasions no evidence of change occurred until one to two hours had passed, and still more often not till twelve to twenty-four hours had elapsed. We do not think that the late reduction could have been due to the action of bacteria or yeast fungi, as we took the greatest pains to prevent their entrance, and there was no sign of decomposition in the mixtures after several days.

We were puzzled to account for the apparently capricious action of the succus entericus. There can be no doubt that the quality and strength of the fluid varied from time to time. We noted that when it had a brown colour its inverting action was more rapid and sure. But this did not occur sufficiently frequently to explain the many cases of failure. We then found that if, in the process of collection, we were careful to add all the mucus found on the sponge to succus entericus, we had no difficulty in obtaining a reduction; but the freer the succus was from mucus, the the more often we failed. To test this more fully we made two observations daily for some time, taking in parallel series succus entericus only with cane-sugar, and mucus with cane-sugar. We found that in the former case, succus entericus free from mucus, no formation of reducing sugar occurred, but in the latter case the results were uniformly positive. Even distilled water in which the mucus had been washed gave a positive result by the formation of reducing sugar on one occasion.

So as to ascertain more on this point, we took some succus entericus previously found to reduce sugar and containing mucus. The mucus was carefully precipitated by adding acetic acid, and the precipitate filtered off. The precipitate of mucus on the filter-paper was removed by washing with water. The watered mucus so obtained was faintly acid, and was divided into two portions, *a* and *b*. The portion *a* was rendered alkaline, and then tested with cane-sugar; this was converted into grape-sugar. The portion *b* was merely neutralised, and on experiment failed to convert cane-sugar into grape-sugar. The filtrate of succus entericus, which had become acid because of the addition of acetic acid in precipitating the mucus from it, was rendered alkaline, but failed to convert cane into grape sugar. As a check experiment we tested nasal mucus quite fresh, and found that it had no powers of converting cane-sugar into grape-sugar, so that we are inclined to think that the ferment is suspended to some extent in the mucus rather than merely dissolved in the more liquid parts of the succus entericus; and not that the mucus in the juice exerts any converting action *per se,* since the neutralised portion of mucus proved a failure; so too did nasal mucus, while the alkalinised portion of mucus, or that in which the chemical reaction was favorable to the ferment, proved a success. One other point is interesting. Two collections were made on November 12th, 13th, 14th, 15th, 16th; that of the morning, collected on a sponge, was found to have no inverting action, while that of the evening, collected in a bottle, was of good fermentative power—probably since the evening collection was purer, and contained more mucus than the morning. Possibly, too, the effect of the digested and absorbed material during the day stimulated a flow of juice rich in fermentative power.

We would suggest that the factors at work in the inverting power were concentration of the fluid (*i.e.* brown colour and high specific gravity), the amount of mucus in the collected fluid, and the time of day. With regard to this last point it would be instructive to observe whether any difference can be detected in the amylolytic powers of fasting saliva or the peptic powers of gastric juice after fasting, and immediately after a good meal, when in the latter case the glands are replenished with blood, and probably contain some of the results of the activity of the secretion.

Lactic acid was formed when the inverted cane-sugar was kept for some time, and the mixture became neutral in three to four days. We do not regard this as distinct evidence of lactic acid fermentation, partly because of Hoppe-Seyler's observation that lactic acid forms in fluids containing cane and grape sugar if the air be excluded, and partly because lactates were found in the juice when secreted. We had not the means of comparing the amount of lactates present in the fresh juice, and in a mixture of cane-sugar and succus entericus some days old.

As to the amount of reducing sugar; this was tested by Pavy's fluid, as it is more convenient than Fehling's solution, since the ammonia dissolves the proteids, and it is easier to detect when a solution loses its colour than when it alters completely from blue to red.

TABLE II.—*Amount of Reducing Sugar formed by Action of Succus Entericus on Cane-sugar.*

Date.	Age of succus entericus used.	Amount of cane-sugar experimented on.	Duration of experiment.	Amount of reducing sugar formed.	Remarks.
Oct. 30	1 day	1 gramme	48 hours	No sugar	
	,,	,,	4 days	·01 gramme	
Nov. 1	Fresh	,,	2 hours	Very small amount	
	,,	,,	3 days	·19 gramme	
,, 3	,,	,,	3 hours	Very small amount	
	,,	,,	3 days	·05 gramme	
,, 13	3 hours	,,	1 hour	No reduction	
	,,	,,	12 hours	·0375 gramme	
	,,	,,	24 ,,	·1 ,,	
	,,	,,	48 ,,	·4 ,,	
	,,	,,	96 ,,	·5 ,,	
,, 14	Fresh	,,	24 ,,	Slight reduction	
	,,	,,	48 ,,	·075 gramme	
	,,	,,	96 ,,	·09 ,,	
,, 16	,,	,,	45 min.	Faint reduction	
	,,	,,	90 ,,	·04 gramme	
	,,	,,	30 hours	·1 ,,	
,, 20	3 hours	,,	30 min.	Slight reduction	
	,,	,,	14 hours	More reduction	
	,,	,,	24 ,,	·012 gramme	
,, 21	11 hours	,,	43 ,,	·01 ,,	Morning, succ. ent.
	2 hours	,,	43 ,,	·016 ,,	Evening, ,,

Experiments on Starch.

These were performed in test-tubes at 37°, in like manner to the cane-sugar trials.

TABLE III.—*Experiments on Starch.*

Substance acted on.	Form of secretion.	Number of experiments in which reduction was obtained.			Number of experiments in which no reduction was obtained.	Total number of experiments.	Remarks.
		In 12 hours and under.	In 24 hours and under.	Over 24 hours.			
Starch	Succus entericus	7	18	15	32	72	2 failures and 4 reductions with unboiled starch.
,,	Succus entericus without mucus	0	2	1	1	4	
,,	Mucus alone	0	1	0	1	2	
Totals . .		7	21	16	34	78	
			44				

It is thus seen that in 78 experiments reduction was obtained on 44 occasions, or 56·5 per cent. were successful. Of the unsuccessful trials one lasted for one hour, and one for three hours, and thus did not afford a satisfactory test.

As to the amount of reducing sugar formed we append a table; 2½ c.c. of succus entericus were added to varying quantities of boiled starch.

TABLE IV.—*Amount of Reducing Sugar formed from Starch.*

Date of experiment.	Duration.	Amount of starch.	Amount of sugar formed.	Remarks.
		Gramme.	Gramme.	
Nov. 1	4 days	·1	·09	Sugar present in 2 hours.
,, 2	4 ,,	·1	·05	
,, 3	3 ,,	·1	·015	
,, 4	40 hours	·1	·001	
	5 days	·1	·018	

TABLE IV (*continued*).

Date of experiment.	Duration.	Amount of starch.	Amount of sugar formed.	Remarks.
		Gramme.	Gramme.	
Nov. 5	24 hours	·1	·0125	
	4 days	·1	·03	
„ 9	48 hours	·1	·015	
„ 12	12 „	·1	·01875	
	24 „	·1	·03	
	4 days	·1	·075	
„ 14	48 hours	·05	·025	
„ 15	30 „	·15	·12	
„ 16	32 „	·1	·024	
	5 days	·1	·035	
„ 17	3 „	·5	·025	
„ 19	50 hours	·1	·02	
„ 20	14 „	·1	·02	
	3 days	·1	·07	
„ 18	72 hours	·1	·025	
„ 21	43 „	·1	·037	

It is evident by comparing Tables II and IV that the fermentative powers as to the comparative quantities converted are greater in the case of starch than in cane-sugar. Evidence of change was obtained with starch on one occasion in one hour, twice in two hours, twice in three hours, and once in five hours, but by no means appeared so rapidly as in the case of cane-sugar, where it took place once in three minutes and once in ten minutes. On 61 occasions specimens from the same collection were allowed to act on both starch and cane-sugar, but only in 32 instances was there a similarity of result; in the remaining 29 either starch or cane-sugar alone was changed into reducing sugar. We are somewhat at a loss to account for this, but we failed to be heedful on one point, viz. to leave the test-tubes for the same length of time in both cases. This was an error on our part, which we realised too late.

It is interesting to note that *erythrodextrin* appeared as an intermediate stage in the conversion of starch; in one experiment we found it at the end of two hours, reducing sugar being already present in some quantity.

The variety of reducing sugar formed was certainly glucose, but this merits further allusion in connection with the power of succus entericus in continuing salivary and pancreatic digestion.

We are inclined to think that the presence of mucus played an important part in the conversion of starch, although we have not the same experimental evidence as in the case of cane-sugar. Starch was on one occasion readily altered by mucus from the sponge.

The whole of the starch was not at any time converted into grape-sugar; in many cases a considerable quantity of erythrodextrin was present. The greatest amount of conversion was four fifths of the starch used, and the longer the time the more starch disappeared. Uffelmann's test for lactic acid was obtained five hours after the commencement of the experiment in one case, although the mixture was then neutral in reaction.

Succus Entericus continues Salivary and Pancreatic Digestion of Carbo-hydrates from Maltose to Glucose.

We made some attempts to find out what kind of sugar was formed in intestinal digestion, and for this purpose took advantage of the phenyl-hydrazin test for sugar, which gives with maltose crystals of phenyl maltosazone, and with glucose those of phenyl glucosazone, the two being widely different in appearance under the microscope. The method of procedure was to take a test-tube containing some of the products of digestion, add a little phenyl-hydrazin hydrochloride and sodium acetate in the proportion of two parts of the former to three of the latter, and keep the whole at 100° C. by means of a water-bath for one hour. We did not in any case fail to obtain characteristic crystals. The products of the action of succus entericus on cane-sugar of December 4th and 7th, and starch of December 4th, 7th, and 10th, were tested in this way, and in every case a large amount of phenyl glucosazone was found, with *no trace* of maltosazone.

In each of these cases the digestion process had been going on for a considerable time—in one case twenty-eight hours—and in order to show that even if maltose is formed during intestinal digestion, which is very improbable, since no signs of maltosazone crystals were found amongst those of glucosazone, it is nearly all ultimately converted into glucose, we carried out the following series of experiments.

Exp. 1.—A solution of maltose was taken and divided into three equal parts, A, B, and C. To A were added 5 c.c. of succus entericus three days old, to B 5 c.c. of succus entericus twenty-four hours old, and to C 5 c.c. of distilled water. At the end of fifty-six hours they were all three tested by the phenyl-hydrazin process, and whilst C gave phenyl maltosazone crystals and no trace of glucosazone, both A and B gave abundance of the latter and only traces of the former.

Exp. 2.—A solution of maltose was divided into two parts, A and B; to A 5 c.c. of secretion ten hours old were added, and to B 5 c.c. of water. At the end of forty-eight hours A gave abundance of glucosazone crystals, whilst B gave nothing but maltosazone.

Exp. 3.—Exactly similar to No. 2, but fresh succus was used, and only acted for thirty hours; the result was the same.

Exp. 4.—Similar in every way to the last.

We think that these experiments, which are so uniform in result, go a long way towards proving that maltose and probably any other kind of sugar formed by salivary and pancreatic digestion are ultimately converted into glucose by the succus entericus before absorption by the small intestine.

Isolation of Ferments.

We endeavoured on several occasions, by precipitating the proteids of the juice with absolute alcohol and then extracting with glycerine, to isolate the amylolytic ferment, but without much success. The following is an account of the experiments which we made in this direction.

No. 1.—Succus entericus of September 20th, with three times its volume of absolute alcohol, was allowed to stand twenty-three days, then filtered, and the filter-paper with the precipitate upon it placed in glycerine.

This extract had no effect on either cane-sugar or boiled starch solutions.

No. 2.—Secretion of September 24th, 27th, 28th, and

30th allowed to stand for eight days with three to four volumes of absolute alcohol, then filtered. Filter-paper dried and placed in glycerine.

This extract caused reduction to appear in a boiled starch solution in twenty-four hours, but had no effect on cane-sugar.

No. 3.—About 60 c.c. of secretion which had accumulated during the previous six days were added to 200 c.c. of absolute alcohol on November 6th, and allowed to stand till the following January 7th (eight weeks). This precipitate was treated as before, but when it had been soaking in glycerine for some weeks it was taken out and rubbed up in some more glycerine.

The first portion of glycerine had no action on either sugar or starch; the second had also no action on sugar, but converted starch to a slight extent in three days.

No. 4.—A similar experiment, 55 c.c. of secretion being placed aside with 165 c.c. of alcohol for five weeks. On this occasion also the filter-paper was squeezed out in a second portion of glycerine after standing for some time in the first portion; the latter again had no action on starch or sugar, whilst the former only acted very slowly (three days) on starch, and not at all on sugar.

In the cases of Nos. 3 and 4 both extracts were tried with maltose by the phenyl-hydrazin method, but with these no formation of glucose was found to take place.

No. 5.—Secretion of December 7th was placed aside in three times its volume of alcohol for a month, and then extracted with glycerine. This, however, acted on neither starch, cane-sugar, nor maltose.

It will thus be seen that it is possible to obtain a ferment from the secretion of the small intestine which will convert starch into glucose (the presence of glucose was shown by the phenyl-hydrazin test in the case of Nos. 3 and 4), but for some reason we failed to isolate any ferment which would act in a similar way upon cane-sugar. Nos. 1, 2, 3, and 4 were also tested with albumen in neutral and alkaline solutions.

A solution of egg-albumen was made, and divided into five equal parts; to one glycerine was added, and to each of the others some extract. The mixtures were then placed in dialysers, and kept at 37° C., and at the end of thirty-six hours peptones were not found in the water of dialysation in any case.

Another solution was taken, and made alkaline with Na_2CO_3. This was treated in the same way with a like result.

A similar experiment was carried out with gelatin, but the liquids were placed in test-tubes instead of dialysers. It was then found that at the end of thirty-six hours they would none of them gelatinise.

When at his final operation Mr. Davies-Colley closed the fistula, the mucous membrane which was then dissected away was at once placed in a bottle containing alcohol and water in equal proportions. Some small pieces were cut off for microscopical examination, but otherwise the membrane remained undisturbed in the bottle until the beginning of April. It was then divided into two parts, one part being placed in glycerine and water (50 per cent. of each), and the other half ground up with powdered glass in a mortar, and then placed in glycerine and water.

It was found that the glycerine in which the undamaged mucous membrane had been soaked had no action on cane-sugar or maltose, and only a very faint action on starch; whilst the mixture of finely-divided membrane, glycerine, and water converted all three into glucose, the time of its action being twenty-four hours in the case of starch, thirty-six in that of cane-sugar, and twelve hours in that of maltose.

These two extracts also both acted on gelatine, preventing its coagulation on cooling, which glycerine did not; and no formation of peptones from egg-albumen, neutral or alkaline, could be obtained in either case.

Experiments on Milk.

Exp. 1 (December 7th).—5 c.c. of succus entericus of December 6th collection, neutral in reaction, were added to 5 c.c. of fresh milk. In one hour a flocculent precipitate fell.

Exp. 2 (December 7th).—5 c.c. of fresh succus entericus added to 5 c.c. of fresh milk; in one hour a flocculent precipitate came down. We were unable to determine if this were casein, but we think it probable.

Exp. 3 (December 10th).—Two test-tubes, in one (A) 5 c.c. of milk placed, in the other (B) 5 c.c. of milk and 5 c.c. of fresh succus. In two hours both had a thick layer of creamy appearance on the surface, and both were neutral. At the end of twenty-four hours the milk alone (A) had not changed in appearance, although somewhat acid, while the tube (B) containing the mixture presented a striking difference. On the top of the mixture was a layer similar to that in (A), but twice as thick; below this thick layer was a slightly *turbid* acid fluid equal to three fourths of the total liquid first placed in the tube. It seemed as if by the action of the succus a separation into curds and whey had occurred, and we think it possible that succus entericus contains a ferment capable of precipitating caseinogen.

Experiments on Absorption by the Bowel.

We made some attempts to investigate the absorbent qualities of the small piece of mucous membrane under observation, but met with so much difficulty in securing the retention of even such small quantities of liquid as 2 to 3 c.c., that we were prevented from carrying out any complete course of experiments. Any attempt to keep solid matter, such as pieces of egg-albumen, inside the fistula always failed, owing to the vascularity of the mucous membrane, which quite filled the lumen of the intestine; the intra-abdominal pressure, which, as we shall point out, was considerable; and probably to some peristalsis set up by irritation in the isolated piece of intestine.

The following experiments were made:

No. 1 (November 28th).—A small piece of muslin containing a considerable quantity of starch in its texture was inserted in the form of a bag enclosing some unboiled isinglass. It was found at the end of twenty-four hours lying on the sponge; there was not sufficient loss of weight to show much action on the gelatine, but the muslin, which originally gave a deep blue with iodine solution, now gave a violet colour, very suggestive of erythrodextrin.

No. 2 (November 11th).—2 to 3 c.c. of a 1 per cent. solution of boiled starch were injected by means of a thoroughly sterilised syringe, the nozzle of which was passed into the fistula as far as possible. At the end of five minutes the syringe was again inserted, and as much as possible withdrawn. Only a few drops (½ c.c. ?) could be obtained, and this would not reduce Liq. Pavii. On the addition of Liq. Iodi there was no coloration except of some small pieces of mucus which were floating about, and which acquired a mauve tint (erythrodextrin ?). There was no reducing sugar in the secretion of the following twenty-four hours.

No. 3 (December 6th).—3 to 4 c.c. of a strong boiled solution of starch were inserted. At the end of twenty-four hours there was no trace of starch or sugar to be found in (i) the secretion of that period, (ii) the drop or two of liquid which could be drawn off by means of the syringe, or (iii) the boiled water with which the bowel was then washed out.

It would seem from these three specimens that boiled starch is quickly converted into sugar and absorbed when placed in immediate contact with the mucous membrane.

No. 4 (October 16th).—A small piece of unboiled starch, with a similar piece of cane-sugar, was inserted. In the secretion of the following twenty-four hours there was no trace of sugar or starch.

No. 5 (November 27th).—A thick paste of starch and peptone rubbed up with distilled water was made, and about 3 c.c. were inserted at 11 a.m., a sponge being applied as usual to the fistula. This sponge was removed at 12 o'clock. The mucus which had collected on the sponge contained some of the paste; it gave a fairly-marked blue with Liquor Iodi, but the sponge and mucus washed in water and dialysed gave no trace of peptone or sugar. A fresh sponge was then put on, and taken off at 6 p.m. The mucus on this sponge also contained some of the paste, but there was no evidence of any of the peptone having escaped absorption, and the whole amount of the mixture which came away was not more than one sixth of that which had been inserted.

Two or three drops of secretion were drawn off by means of the syringe at 6 p.m., and it contained no reducing sugar

—enough could not be obtained to test for peptones. The secretion which had collected in the sponge gave no peptone or sugar reactions.

No. 6 (November 27th).—When the fistula had been thoroughly washed out with boiled water more of the mixture was inserted and the sponge left on till midday of the following day—that is, for eighteen hours. The result was the same as before. Some unchanged starch was found in the mucus on the sponge, but the secretion even on dialysation for three days gave no evidence of peptones or sugar. The fistula was then carefully syringed out with boiled water.

These two experiments point to the complete absorption of peptone and the partial conversion into sugar with absorption of unboiled starch.

In addition to the experiment (No. 4) mentioned above, two other attempts were made to investigate the absorption of cane-sugar.

No. 7 (November 10th).—2½ c.c. of a 10 per cent. solution of cane-sugar were injected. After five minutes as much as possible (two or three drops) was withdrawn by means of the syringe, and was found to reduce Liquor Pavii freely, but there was not enough to admit of an estimation of the amount of grape-sugar present. The secretion of the following twenty-four hours contained no reducing sugar, nor was there any in the water with which the fistula was washed out at the end of that time.

No. 8 (November 29th).—Some small pieces of cane-sugar were inserted. At the end of sixteen hours reducing sugar was found in the sponge, and also in the water with which the fistula was syringed out.

It would, therefore, seem that cane-sugar in contact with the mucous membrane of the small intestine is converted into grape-sugar and absorbed.

Microscopical appearances of the sac.—Portions of the mucous membrane were taken from the external opening of the sac, from the middle of the sac, and from the fundus. Half of each specimen was stained in hæmatoxylin and half in lithium carmine, the former being in every case more successful. The specimen obtained from the mouth of the sac showed the mucous membrane rather thickened with hypertrophied villi. The glandular tubes were patent, but for the most part denuded of epithelium. There was a considerable excess of lymphatic substance everywhere, with large and very abundant nuclei, whilst in the submucosa there was some formation of fibrous tissue. The outlines of the muscular fibres were obscured, and the individual cells had in many places acquired a granular appearance. The specimen from the middle of the sac showed the outlines of Lieberkühn's follicles to be obscured as well as those of the individual cells. There appeared to have been a rapid proliferation of the cells, together with some degeneration of a hyaline nature, causing blocking of the glandular tubes, distorting their outlines, and rendering them somewhat indefinite. The muscle-fibres were abundant and well stained, the villi of normal size, and the supporting tissue of the mucous membrane somewhat hypertrophied, but not to such an extent as at the mouth of the diverticulum.

The third specimen, from the fundus, was very little different from that of normal mucous membrane of that part of the small intestine. The outlines of the gland-cells were slightly obscured, but the regular appearance of the cells lining the tubes was evident. The villi were of normal size, the supporting tissue of the mucous membrane not in excess, and the muscle-fibres more distinct than at the mouth of the sac.

Experiments on Intra-abdominal Pressure.

These were carried out by means of an apparatus similar to a cardiac sound, but containing water instead of oil or mercury, and connected with a water manometer. The bulb of the sound was passed empty through the fistula and then dilated with water, and the mouth of the fistula was closed as far as possible on the tube. We noted the effects of the movements of respiration in altering the intra-abdominal pressure.

Exp. 1 (October 17th).—In ordinary expiration a negative pressure of 1 to 2 cm. of water appeared, and in ordinary inspiration the pressure was increased by 3 cm. of water.

In forced expiration the pressure was greatly increased, the manometer rising 15 cm. of water.

Exp. 2 (October 26th).—In both forced inspiration and expiration the intra-abdominal pressure was much increased, in the former 9 cm., and in the latter 10 cm. of water. In ordinary inspiration the pressure rose 2 cm., and in ordinary expiration it fell 3 cm. of water.

ON THE VOLUMETRIC DETERMINATION OF URIC ACID IN URINE.

By F. GOWLAND HOPKINS, B.Sc., F.I.C.,
GULL RESEARCH STUDENT.

In a paper read before the Physiological Society in March last I described a method by which uric acid may be rapidly and completely separated from the urine in the form of ammonium urate. This result is obtained by the *complete saturation* of the urine with ammonium chloride, whereby the uric acid is precipitated in the form of ammonium urate,—not, as in Fokker's well-known process,[1] slowly and incompletely, but quantitatively, and within a period of two hours at most.

By means of this separation the uric acid may be estimated gravimetrically with very great ease and accuracy, since the free acid may be at once liberated from the ammonium compound by treatment with mineral acids, and so weighed direct. Such a process I have elsewhere described.[2] But the uric acid, being thus separated from the other urinary constituents without the inconvenient presence of any heavy metal such as silver, would seem to be in a condition for direct volumetric estimation by any method which is applicable to pure urates.

The only methods hitherto suggested for the direct titra-

[1] Salkowski, 'Fresenius's Zeitsch.,' xvi, 371.

[2] I have described the gravimetric method, and fully discussed the preliminary precipitation by means of ammonium chloride, in a paper shortly to be presented to the Royal Society. I do not, therefore, enter into any detail here.

tion of uric acid itself, as distinct from indirect estimations by titration of a combined base, are (1) the use of permanganate solutions (*vide* Sutton, 'Volumetric Analysis,' fourth edition, p. 306, where the author suggests an inadequate preliminary separation by means of HCl), and (2) estimation by means of ammoniacal cupric solution or by Fehling's solution (Pavy, 'Med.-Chir. Trans.,' vol. lxiii).

I have carefully examined these two methods, with the object of applying them directly to the ammonium urate precipitate produced by saturation with ammonium chloride.

Of the two the permanganate method has alone yielded results of any value in my hands. These results will be first described.

A. *Titration of the Ammonium Urate Precipitate by means of Potassium Permanganate.*

Reagents required :

(*a*) Pure ammonium chloride, finely powdered. Ammonium chloride, commonly sold as pure, being prepared from the ammoniacal liquor of gas-works, frequently contains organic matter; if possible a salt should be obtained which does not blacken on heating.

(*b*) A saturated solution of ammonium sulphate,[1] for washing the precipitated urate.

(*c*) A twentieth-normal solution of permanganate of potash, prepared by dissolving 1·578 grammes of the salt in one litre of distilled water.

(*d*) Pure strong sulphuric acid.

I. To 100 c.c. of the urine solid ammonium chloride is added to saturation, from 30 to 35 grammes being required. After the addition of the salt the sample is allowed to stand for two hours. The high specific gravity of such a saturated solution causes a tendency in the resulting precipitate to float at the top; occasional stirring or shaking will, however, secure its subsidence.

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[1] In all other processes based upon the separation described above, a saturated solution of ammonium chloride itself is best employed for washing; but for the subsequent use of permanganate it is necessary to get rid of chlorides. Washing with the sulphate is slow, and the pump may be used with advantage, but it is by no means necessary.

II. The precipitated urate is now filtered off through thin filter-paper. It will be found to filter with great ease, especially if the precipitate has been previously caused to sink by stirring.

III. The precipitate is now washed twice or thrice on the filter with the saturated $(NH_4)_2SO_4$ solution, and finally washed into a beaker by means of a jet of hot distilled water. It is then completely dissolved by warming with the addition of a few drops of Na_2CO_3 solution.

IV. After thorough cooling the solution is made up to exactly 100 c.c., and then transferred to a flask of about 250 c.c. capacity; 20 c.c. of strong sulphuric acid are now rapidly added, the mixture well shaken, and titration with the permanganate *immediately* performed. The latter is run in from a graduated burette, quickly at first, but more slowly towards the end of the reaction, until a faint permanent pink colour is obtained. The amount of permanganate used is then read off, each cubic centimetre being equal to ·00375 grm. uric acid.

The oxidation of uric acid by means of permanganate of potash proceeds, on standing, greatly beyond the stage thus obtained; but, under the exact conditions described above, the close of a definite reaction is registered by the first appearance of permanent pink colour, and, as stated above, each cubic centimetre of permanganate is then equal to exactly ·00375 grm. of uric acid. This colour will afterwards fade, but this need not concern the operator. I have confirmed the above results by repeated experiments on pure uric acid.

It must, however, be carefully observed that the experiments are to be conducted under the precise conditions, with regard to dilution and the like, described above. The temperature of titration, for instance, is important in ensuring a determinate reaction, the addition of 20 c.c. of sulphuric acid to 100 c.c. of cold aqueous solution producing just such a temperature (about 60° C.) as is necessary; hence the necessity of using exact proportions, and of proceeding with the titration directly the acid is added.

The ammonium urate as precipitated by means of my process is always slightly coloured, but any organic impurities present are so small in amount as scarcely to affect the result with permanganate.

B. *Titration by means of Cupric Solutions.*

Dr. Pavy, in a communication to which reference has been given above, suggested a method for the estimation of uric acid in urine by means of his ammoniacal cupric solution. The total reducing power of the original sample was to be first determined, and then the residual reducing power after removal of the uric acid by means of lead acetate, the difference showing the quantity of the uric acid present.

In the hands of subsequent observers this process has proved a difficult if not an impossible one. Personally I have been unable to determine a definite reduction factor even when working with pure urates, though I have performed a large number of experiments, varying the conditions in every possible way.

In the first place, the reduction of copper by uric acid is an extremely slow process when compared with the action of sugars; so slow, indeed, that, when working with the ammoniacal cupric solution as usually prepared, all the ammonia is inevitably boiled away before the completion of the process.

To obviate this a second burette was coupled on to the boiling flask, and from it a fresh supply of strong ammonia was from time to time added. This plan prevented the precipitation of oxide of copper, but did not obviate the next difficulty which presented itself, namely, that before a true end-point was reached, a yellow colour developed, independently of the separation of oxide.

I found, finally, that by adding, before beginning the experiment, 25 c.c. of a saturated solution of AmCl to each 10 c.c. of blue liquid in the flask, each of these difficulties could be overcome. However long the solution was now boiled no precipitation occurred, and little or no yellow colour developed. Such a course as this, on the other hand, completely alters the conditions of experiment, because, as the caustic alkali in the blue liquid gradually replaces the ammonia of the AmCl when the solution is boiled, it becomes gradually neutralised, and all observers agree that the percentage of caustic alkali present profoundly affects the reduction factor of Pavy's solution.

What finally led me to abandon the process, however, was the great importance of the time element in titration, which became evident when ammonium chloride was added as above described. For instance, if the uric acid solution was added to the boiling copper solution at about the normal rate (such as one would use in estimating sugars), about 15 mgrms. (15 c.c. of a ·1 per cent. solution) of uric acid appeared to be necessary to decolourise 10 c.c.;[1] but if, on the other hand, only 8 mgrms. were added, and the flask boiled for five minutes without further addition, decolorisation occurred equally well.

It seems to me, therefore, very difficult, if not impossible, to define conditions which shall determine a simple molecular reduction of copper by uric acid.

c. *Titration by an Alkalimetric Method.*

Investigation proves that in the precipitate produced in my process by saturating urine with AmCl, the uric acid exists definitely combined as the acid ammonium salt. As far as my experience has at present gone this always obtains.[2]

This being the case, the possibility of estimating the uric acid indirectly by an ordinary alkalimetric process at once suggests itself, the only requirement being an indicator sensitive to mineral acids, but unaffected by uric acid.

The only substance that I have hitherto been able to obtain exhibiting such properties satisfactorily is *methyl orange*, an indicator which has of late come largely into favour in analytical laboratories.[3] The body is yellow in

[1] 9·33 mgrms. would be requisite according to Dr. Pavy's determination

[2] Although the normal ammonium salt does not exist, Maly has described compounds of the form (*a*) $C_5H_3N_4O_3(NH_4) + C_5H_2N_4O_3(NH_4)_2$ and

(*b*) $2C_5H_3N_4O_3(NH_4) + C_5H_2N_4O_3(NH_4)_2$.

In the presence of a large quantity of AmCl one might expect that such would be formed, but I have never fonnd this to be the case.

[3] I am indebted to Mr. Norman Leonard, B.Sc., for the suggestion that methyl orange would probably serve my purpose here.

alkaline solutions, and pink when free mineral acid is present, but it is quite inert with uric acid.

If, therefore, an alkaline salt of the latter be decomposed by warming with a known amount of standard sulphuric acid, the excess of the latter may be titrated back by standard alkali if methyl orange be used as the indicator, since the neutral point will not be affected by the liberated uric acid.

The above facts are utilised in the following process.

Alkalimetric Method.

Solutions, &c., required :

(*a*) Standard sulphuric acid, one twentieth normal in strength, made by diluting accurately adjusted normal acid.

(*b*) One twentieth normal soda, adjusted to the acid by means of methyl orange.

(*c*) A ·1 per cent. solution of methyl orange.

(*d*) Ammonium chloride, powdered as before, and in saturated solution for washing the precipitate.

200 c.c. of urine are necessary for accuracy in this process. The ammonium chloride is to be added in proportionate quantity exactly as described for the permanganate process above. The first three steps, in fact, are to be carried out precisely as there described, using, however, the chloride instead of the sulphate for washing; but instead of dissolving the ammonium urate in Na_2CO_3 solution one proceeds as follows :

IV. To the urate in the beaker a measured quantity (30 to 40 c.c.) of the standard acid is added, and the whole boiled for some minutes until decomposition is complete; the change from the amorphous urate to the crystalline uric acid being quite obvious.

V. The solution is now thoroughly cooled, diluted to about 200 c.c., a very small quantity (about one drop only) of methyl orange added, and, finally, standard alkali run in till the solution is neutral, the point being indicated by the change of colour from pink to yellow.

The difference between the amount of acid originally added, and the amount of alkali finally required, measures the quantity of the former which has been neutralised by ammonia previously existing as urate of ammonia. Each cubic centimetre of the acid so neutralised is equal to ·0084 grm. uric acid.

As I have said, methyl orange is greatly praised as an indicator by many chemists. Personally I at first found the neutral point rather difficult to define, and it was only after some little practice that I was able to attain to accuracy in its use. The colour due to the slight amount of pigment separated with the ammonium urate adds somewhat to the difficulty of titration, but this is obviated by diluting the solution to 200 c.c. or more before making the estimation. The titration should be performed in a good light.

The following figures will indicate the degree of accuracy which the process has yielded in my hands :

Sample.	Titrated by the alkalimetric method. Milligrammes per 100 c.c.	Weighed. Milligrammes per 100 c.c.
No. 1	72	70
2	60·5	59
3	51	49·5
4	42	42
5	39	36
6	37	38

Summary.

I am satisfied that the separation of uric acid from urine by saturation with AmCl is a process of absolute accuracy, and that gravimetric estimations based upon it may be made equally accurate. A fully satisfactory volumetric process is, doubtless, yet to seek. But of the two processes above described I can confidently recommend the permanganate method as suitable for clinical purposes. It has the disadvantage of requiring the removal of chlorides before the titration is carried out. The fact that the colour marking the end-point afterwards disappears offers no difficulties in practice, the point being quite definite. Experiments with pure urates prove its absolute accuracy. The small amount of organic matter carried down with the ammonium urate, and which acts upon the permanganate, is, as I have said, so slight in amount that it may be safely neglected in clinical work.

In laboratory work the alkalimetric method last described may be preferred by some, and it is especially suitable where a large number of determinations have to be made. The only requisite is a certain amount of preliminary practice in titration. The alkalimetric method has advantages when albuminous and diabetic urines are dealt with, while it is less suitable for alkaline samples.

I have had considerable experience with Haycraft's method, and though quite unable to understand the extraordinarily wide variations from the truth which some observers, especially in England, have ascribed to it, I have nevertheless found it subject to variable error. The permanganate process, though yielding results which are slightly high, is free from this element of variability, and is, therefore, more suited to clinical purposes.

NOTE ON A FORM OF BLOOD-PRESSURE MANOMETER.

By ERNEST H. STARLING, M.D.,

AND

W. M. BAYLISS, B.A., B.Sc.

Since the classical work of Marey on the subject of the pulse and endocardiac pressure-curves, our ideas on the explanation of these have been thrown into a complete state of confusion by the work of later observers. Although the dicrotic notch is still regarded by most authorities as being due to closure of the aortic valves, all possible explanations have been given of the origin of the other secondary waves in the pulse-curve. These latter are, according to some authors, centripetal, and according to others centrifugal in their course. M. von Frey, in a recent work, has made use of modifications of Fick's spring manometer to elucidate this question, and has come to the conclusion that many of the waves on the pulse-curve are reflected waves. According to this observer, the endocardiac pressure-curve during ventricular systole is absolutely free from all secondary vibrations, and hence all secondary waves appearing on the pulse-curve before the closure of the aortic valves must be peripheral in origin.

The whole question of the interpretation of pulse-curves depends on the efficiency of the instruments employed, and on our ability to distinguish in the curve the elements that are instrumental in origin from those which are due to changes in pressure taking place in the part of the vascular system under observation. The first point, therefore, in an investigation on the pulse-curve is to secure a good instrument.

The requirements of a good manometer are as follows:

1. The mass to be moved must be as small as possible.

2. The instrument must be quick, that is to say, it must follow immediately very rapid changes of pressure.

3. It must be as far as possible aperiodic. This may be secured in a blood-pressure manometer by making the periodicity of the instrument so rapid that no instrumental vibration can be mistaken for the slower waves on the pulse-curve.

These requirements are fairly fulfilled by the instrument with the aid of which the accompanying curves were taken. It consists simply of a wide glass tube, drawn out at the end into a very fine capillary tube, with approximately parallel sides, and of which the end is sealed. Before closing the end the tube is filled with water, with the exception of a small air-space about 1·5 cm. long at the end of the capillary. The wide end of the tube is put into connection by means of a short piece of lead tubing with the artery or with the endocardiac cannula. Each rise of pressure in the artery causes compression of the air at the end of the capillary tube, and a rise of the fluid in this tube. A magnified image of the capillary tube with the contained fluid is thrown, by means of a limelight projection system, upon a slit, immediately behind which is a revolving drum covered with sensitive paper. A photograph is thus obtained of the pulse or endocardiac pressure-curve.

Although this instrument is not suited for comparative estimation of blood-pressures, or for giving an exact determination of the actual blood-pressure (since the air becomes gradually expanded by the heat of the lamp used, the zero point does not remain constant), yet the small mass of fluid moved, the freedom from instrumental vibrations, and its rapidity render it perhaps the most perfect form of manometer for recording the true form of the pulse-curve; and in this way it may be used for checking other manometers, and it enables us to judge of their efficiency from the shape of the pulse-curve recorded by them. Other advantages of this instrument are that there is no friction between a recording lever and a blackened surface, and the ordinates are all straight lines, and not curved as in cases where the lever is used. It is, moreover, so simple in construction that it may be made by any one in a few minutes.

On comparing the pulse-curves obtained by means of this instrument (Plate V) with those given by Huertle it will be seen that they correspond in every particular. Our endocardiac pressure-curves are also similar to those of Huertle. On testing Huertle's manometer, we found that we could entirely corroborate his opinion on the merits of his instrument. We have also tested von Frey's instrument, and found the same faults in it that Huertle has pointed out, viz. that the mass of fluid moved is too large, and that the air transmission employed so damps the movement that this form of instrument is very ill adapted for informing us as to the true course of the variations in the blood-pressure at any part of the vascular system.

Soon after we had obtained these curves with our capillary manometer, Huertle's admirable paper on the production of the secondary waves of the pulse-curve appeared. Feeling that we could add nothing to the points which this author, with a clear insight into the mechanical principles involved, had so ably worked out, we discontinued our researches in this direction; and merely publish these few curves as a confirmation of the results obtained by him.

We may note that the work of this observer almost entirely confirms the account of the pulse-curve given by Marey. The secondary elevations in the pulse-curve may be divided into systolic and diastolic. The systolic elevations are those on the curve between the beginning of the rise in pressure and the dicrotic notch. During this period the aortic valves are open, and blood is flowing from the ventricle into the aorta. They are all centrifugal in direction, and starting from the beginning of the arterial system (ventricle and aorta). Simultaneous tracings of endocardiac and aortic pressures are almost identical in form during this part of the curve.

The diastolic waves are those occurring on the curve between the dicrotic notch and the succeeding pulse-wave. The chief of these is the dicrotic elevation which is dependent on the closure of the aortic valves. During the whole of this period the aortic valves are therefore closed, and the intra-arterial pressure is unaffected by intra-cardial events.

DESCRIPTION OF PLATE V,

Illustrating Dr. Starling's and Mr. Bayliss's Note on a Form of Blood-pressure Manometer.

All the curves were taken by the capillary manometer, which was connected with the carotid artery of a medium-sized dog. The animal was anæsthetised with a large dose of morphia and a little chloroform.

Fig. 1.—After division of right vagus (heart-beat still slowed through action of morphia on vagus centre).

Fig. 2.—Both vagi divided. The heart was beating faster and more vigorously.

Fig. 3.—Both vagi divided, and right vagus stimulated with currents of moderate intensity.

The time marking in each case = $\frac{1}{100}$ second. Each marking in the figures contains fifty time markings.

FIG. 1.

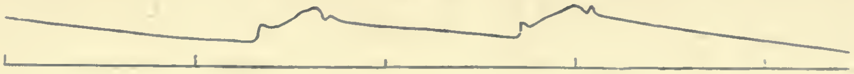


FIG. 2.

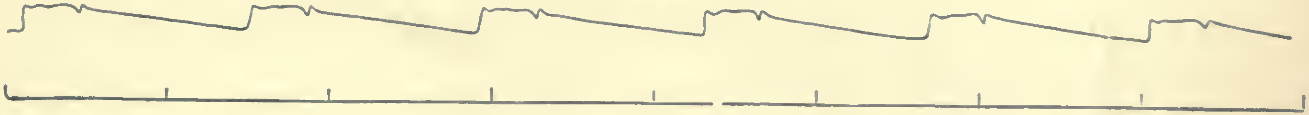
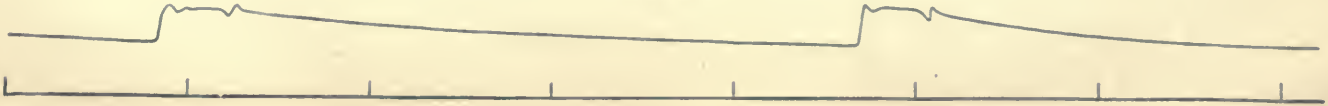


FIG. 3.



A CASE

OF

ACTINOMYCOSIS OF THE LIVER AND LUNGS.

By FREDERICK TAYLOR, M.D.

Henry L—, æt. 42, an engraver, was admitted under my care on July 16th, 1890, complaining of pain in the right side, wasting, constipation, and night sweats. He has always lived in London, and has worked usually from 8 a.m. to 6 p.m., sometimes later. He has had good food, and has not taken stimulants for the last eight months.

Good family history; his father died of bronchitis; his mother and four sisters are alive and healthy.

He suffered a good deal from indigestion before his present illness, and he had an attack of colic a fortnight before Christmas, 1889. Has "winter cough." Has been much constipated since the commencement of the present year, and has taken opening medicine.

Present illness began about six weeks ago, but he had been feeling languid and out of sorts for three months previously. Six weeks ago he noticed a pain in the right groin which did not give him much trouble, but a fortnight later he observed a swelling in the above situation. The pain was not noticeable at times, at others it was intense and shooting in character. He has only had his bowels open when taking purgatives, and the motions have then been loose; but there has been no special pain on going to stool. He gave up work six weeks ago, but did not take to his bed. His appetite has been fairly good, and he has not been sick. He has never passed blood with his urine, and has never had any pain or difficulty with micturition.

On admission the patient is much emaciated, and has a flushed, anxious face.

The abdomen is somewhat distended, and the abdominal muscles are very rigid. On deep pressure in the right flank, just above and anterior to the right anterior superior iliac spine, the patient experiences a sharp pain, and a mass can be felt which is rather firmly fixed, and does not move away from under the finger. On bimanual examination it is recognised that a tumour occupies the right flank, extending from the right costal margin nearly to the iliac crest; internally it does not pass beyond the vertical line dropped from the nipple; backwards it reaches the lumbar region; its surface is moderately smooth, or at any rate not markedly lobulated. It is firm, resistant; and at that time I could not detect any fluctuation. It descended very little during inspiration, but it did not appear to me to involve the abdominal walls. The hepatic dulness begins at the upper border of the sixth rib, and reaches internal to the nipple somewhat lower than usual, while external to the nipple line it is continuous with dulness over the tumour.

The lungs appeared to be normal; the right base seemed to be quite free. Respirations 24. The heart's impulse was in the fifth space, one inch internal to the nipple. There was a slight apical systolic murmur. Pulse 102, hard.

The urine, examined on the 19th, is of medium colour, sp. gr. 1022; gives a slight cloudy deposit, and contains neither albumen, sugar, nor blood.

The temperature was 99·6° on the morning after admission, 90·8° the following morning, and 101·2° in the evening. During the next fortnight or more the temperature was close upon 100° every morning, and above or below 102° each evening.

The opinion at first expressed was that the swelling was connected with the kidney, and the persistent pyrexia suggested that it was inflammatory, and that it might be a tubercular kidney; but the perfectly normal condition of the urine presented a difficulty.

During the rest of the month of July there was no great change in the condition of the patient. He continued to suffer pain in the tumour, he was markedly ill, continued to lose flesh so far as could be judged without scales, had night sweats and the remittent pyrexia above mentioned. The tumour became somewhat larger, and then yielded distinct fluctuation. The blood examined on August 1st showed no excess of white corpuscles, the red corpuscles 91 per cent. of the normal.

Mr. Jacobson was asked to see the patient, with a view to exploration, and this operation was performed by him on August 5th under chloroform.

An incision was made five inches in length below and in the direction of the last rib. On cutting through the lumbar fascia a quantity of foul pus made its escape. A Key's director was used to finish the necessary division of the fascia, and free hæmorrhage took place at this time. The cavity was limited in extent, and quite separate from another lump which could be felt just behind; the former appeared certainly to be outside the kidney. The posterior swelling was then punctured with a trocar and cannula, and was found to contain pus, the egress of which was facilitated by the use of dressing forceps. The cavity now reached appeared to be in the kidney; it was irregular, loculated, and presented some resemblance to the interior of the dilated pelvis of a kidney. A drainage-tube was inserted into each cavity; strips of blue gauze were used as plugs, and the whole was covered by blue strips and a gauze bandage.

The pus from the kidney was examined both for crystals and for tubercle bacilli, but none were found.

On the evening after the operation the temperature fell to 97·2°, and was 97·8° the next morning. But it rose to 99° again in the evening, and for the next fortnight oscillated between 99° and 101°, with occasional evening rises to 102° and 103°.

August 6th.—The patient is fairly comfortable, but did not sleep much. When the wound was dressed, it was not abundant but very foul. Hot boracic dressings were employed.

7th.—The urine was thick with urates and mucus, of sp. gr. 1030, free from albumen. The wound was again dressed, and the drainage-tubes were removed and replaced.

8th.—The urine is of light sherry colour, with a mucous cloud. It contains no albumen, but under the microscope are seen pus-cells, epithelium-cells, and (?) spindle-cells. The wound is less foul.

10th.—The wound is quite sweet; boracic dressings discontinued.

11th.—Urine sp. gr. 1020; slightly alkaline, foul fishy odour; a trace of albumen; no sugar nor blood; under the microscope a few pus-cells, epithelial cells, débris, and bacteria; urea 2·2 per cent. Edges of wound look healthy; discharge sweet, consisting of bloody serum. Tongue fairly clean, appetite good, sleeps better. Ordered 15 gr. of ammonium benzoate in infusion of buchu, three times daily. Farinaceous diet.

16th.—Urine is alkaline, contains a trace of albumen, and deposits phosphates. Ordered benzoic acid, salicylic acid, and borax, 10 gr. of each in water every four hours.

17th.—The bladder was washed out with a solution of borax in glycerine and water (Thompson's fluid). The wound looks well.

19th.—The urine is faintly alkaline, but not so foul. The patient sleeps fairly, but the appetite is poor and the bowels are troublesome as usual.

20th.—There was delirium last night. During the next few days there was no material change. On the 29th he is again reported to have been delirious in the night, and not perhaps quite clear-headed in the morning. On examining the chest, the heart sounds could be heard all over it; and there were some mucous râles at the right apex. Pulse 114; resp. 20. He was more emaciated, the cheeks flushed, and eyes bright. He was delirious again in the evening, and he died rather suddenly at a quarter to 6 a.m. on September 1st. Throughout the case the temperature continued to oscillate between 98° or 99° in the morning, and 101° or 102° in the evening.

The inspection was made on the same day by Dr. Perry.

The operation wound was closed except for the round opening through which the drainage-tube passed. A finger introduced into this aperture passed through a considerable thickness of inflammatory tissue into a cavity with flaccid walls containing a little pus. When pushed in as far as it would go the finger came in contact with a soft substance, which proved to be the disintegrated liver. The boundaries of the cavities were as follows:—Above was the right kidney firmly adherent to the under surface of the liver, and separated from the cavity by a thick layer of fibroid tissues; in front were the liver and ascending colon, and below the ascending colon and cæcum, all matted together by inflammatory tissue, thus forming a space separated from the general cavity of the peritoneum. The inflammation, however, had extended from this sac to the general peritoneum, and there was a considerable quantity of yellowish lymph on the intestine, which was somewhat distended, while its coat was œdematous. The amount of lymph in the pelvis was considerable. The edge of the liver was about two inches below the margin of the costal arch. On inspection of its convex surface a number of small white nodules were seen beneath the capsule, looking like miliary tubercles, and a large yellowish-white patch, formed by aggregation of these nodules, was observed in the substance of the liver above the fundus of the gall-bladder. The right lobe was adherent laterally to the abdominal wall, and in removing it the abscess cavity above described was opened. A horizontal section through the right lobe showed it to be in a condition of purulent infiltration, the proper liver substance having disappeared and the fibrous framework alone being left, so that the appearance was that of a reticulum, the meshes of which were full of pus. The colour of the pus was yellowish white, and in it could be distinguished very minute specks, whiter than the fluid in which they were immersed. Microscopically these specks were found to consist of the ray fungus. The growth in the liver had a lobulated margin; at this part it was more solid and caseous-looking than in the more central portions. The colour was yellowish white.

In addition to the main disease of the liver mentioned above there were here and there little white grains in it, looking very like miliary tubercles. The microscope showed that these consisted of aggregations of small round-cells, in which could be seen the clubs and central granular parts of the fungus.

In the duodenum were two openings, one, one and a half inches from the pylorus, and the other half an inch further on, from which a probe passed into the disintegrated liver substance, the duodenum being adherent to the liver.

The coils of intestine were adherent to each other by recent lymph, and in the pelvis there were several ounces of pus, in which, however, the white grains already described were not seen. The intestines were normal except for œdema of their walls; they were moderately distended. In the ascending colon, as mentioned, there were two sinuses communicating with the cavity of the abscess.

Pleuræ.—There were a few filamentous adhesions at the right apex; the rest of the pleura on both sides was normal.

Lungs.—Both lungs contained scattered throughout them numerous small nodules rather larger than ordinary miliary tubercles. The lung tissue was crepitant; there was a considerable quantity of pus in the smaller tubes. On examination of these nodules they were found to consist of a small white centre, about the size of a pin's head, surrounded by a dark and deeply congested zone. The white material could be squeezed out, being of a curdy consistence, and therefore softer than a miliary tubercle. Microscopically the appearances of each nodule were those of a focus formed by a dense collection of small round-cells, among which were seen the rosettes of the fungus. Surrounding this focus was a zone of extravasated red corpuscles.

Larynx, trachea, bronchi and *bronchial glands,* and *mediastinum* all normal. *Diaphragm* slightly adherent to right lobe of liver. *Pericardium* showed slight œdema of its visceral layer. *Heart,* firmly contracted and normal. *Vessels.*—Pulmonary artery normal, no thrombus. The aorta and coronary arteries were slightly atheromatous.

Kidneys both normal, except that they were a good deal congested.

Mouth, pharynx, œsophagus and *stomach, gall-bladder, pancreas, spleen,* and *supra-renal capsules* normal.

The *jaws* were carefully examined; there was no sign of abscess, caries, or necrosis about the maxillæ; two of the lower molars were carious.

The condition of liver is shown in the accompanying figures, which have been reproduced from photographs of the two sides of the specimen now in the Guy's Hospital Museum. Fig. 1 is a section through the right lobe of the liver, measuring actually 7½ inches by 2½ inches. The mass of diseased tissue in the lower or anterior part of the organ measures 5 inches by 1¾ inches; the line dividing it represents the horizontal incision above mentioned. In the posterior or upper border of the liver are foci of disease, of which the largest three measure respectively three quarters, three eighths, and one quarter of an inch in diameter. The intervening substance of the liver appears to be perfectly healthy.

Fig. 1.

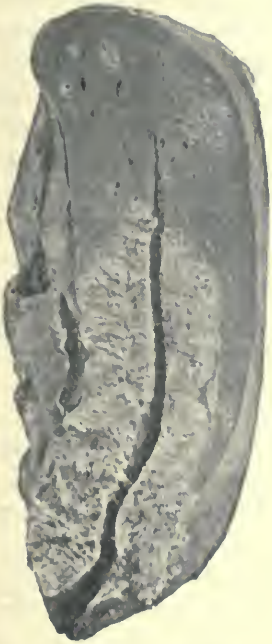


Fig. 2.

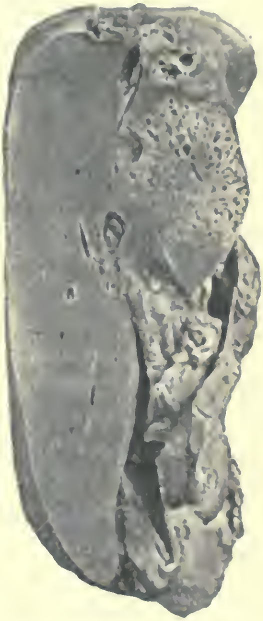


Fig. 2 represents a parallel section through the lobulus

Spigelii, 3½ inches to the left of the section (fig. 1). The large focus of disease in the lobulus Spigelii measures 1½ by 1¾ inches, and the smaller one is half an inch in diameter.

No apology is needed for the publication of this case of actinomycosis, as the cases which have been observed and recorded in this country are not as yet very numerous. At the same time the number of articles and treatises that have been written on the subject will render it unnecessary for me to deal with it at any great length.[1] The essential feature of the disease is the introduction of the Actinomyces or ray fungus into the body, and the production of one or more inflammatory lesions as a direct result of the growth, reproduction, or spread of the fungus.

The ray fungus, which probably belongs to a more highly developed group of bacteria, forms a clump of densely matted mycelial threads, of which those on the periphery of the mass undergo a peculiar form of degeneration. This results in the production of thickened extremities, the well-known clubs, which, lying packed side by side, radiate from the centre of the mass in a characteristic manner. This condition can be recognised both when the fungus is examined in the tissues *in situ*, and in the yellow or green granules which escape in the pus or the secretions.

Since it was known first as occurring in cattle, it was when found in man thought to have been conveyed from the animal to the human system. This idea has, however, been given up, and there can be no doubt that both cattle and human beings acquire it from some common source. In cattle the lesions due to the fungus frequently occur in the tongue or in the neighbourhood of the jaw, from which it would seem highly probable that it is taken in with the food in some way or other, and the explanation which agrees with numerous cases, both in man and animals, is that the fungus is present in some form of corn taken as food by the latter, or occasionally chewed by the former. Boström, in a valuable paper,[2] describes how he has made successive sections of some of the inflammatory lesions, and has actually found in the centre particles of the seed or seed-coats of barley lying amidst a fertile growth of the Actinomyces.

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[1] A very full bibliography is given by Dr. Hodenpyl in the 'New York Medical Record' for December 13th, 1890, p. 656.

[2] Ziegler's 'Beiträge,' 1891, Bd. ix, p. 1.

Thus it seems that the fungus must often be introduced upon solid particles of food taken into the mouth; and that being the case, it must be regarded as a chance whether the fungus first gets a hold in the mouth, in the œsophagus, or in a lower part of the alimentary canal. Its frequent invasion of the lower jaw is probably best explained by its being retained by the teeth, especially decayed teeth, in the process of mastication; but the particles of corn carrying the fungus may penetrate the mucous membrane of the œsophagus, whence they may pass into the mediastinal tissue or towards the spinal column (see Boström's paper); or they may first be arrested in the intestinal wall.

In many cases the lungs have been attacked, and sometimes this invasion seems to have started in the bronchi, as if particles of fungus were really inhaled, and being arrested in the bronchial tubes set up inflammatory changes, which may be of the most extensive kind. In all cases the tendency is for the surrounding tissue to be infiltrated with leucocytes, and ultimately suppuration occurs. But this does not necessarily kill the Actinomyces, for the fungus multiplies, and each fresh colony sets up similar inflammatory lesions, so that eventually—in the neighbourhood of the jaw, for instance, or in the liver—wide-spread suppuration occurs, producing a number of abscesses honeycombing the tissue in a unique manner, and spreading far and wide into adjacent tissues and structures. In the lung, bronchitis, broncho-pneumonia, extensive consolidation, pleurisy, and empyema may occur, and the suppuration may extend outwards, so as to produce abscesses under the skin.

Turning now to the present case, I am afraid there is no evidence as to how the disease was contracted. He was an engineer by occupation, and lived in the south of London. The cause of his disease was not suspected during his life, and my attempts to ascertain from his friends whether he had eaten, or was likely to have eaten, grains of raw barley just prior to his illness were not attended with success.

Boström points out, in confirmation of the view that mastication or ingestion of grains of barley is a frequent means of infection, that in a large proportion of the cases the illness has commenced in the autumn or late summer, when corn is likely to be accessible. According to the history my patient began to be ill in the early part of June, when he is not more likely to have obtained corn than at other periods of the year.

The case presented itself to us as a local inflammation in the right flank, suggesting a perinephritic abscess or tubercular kidney, but it moved scarcely at all in deep inspiration. The skin was not at first implicated, and the urine was quite normal, containing neither albumen nor blood; but he had severe pyrexia, with rigors and profuse sweating. The diagnosis was, therefore, uncertain. Shortly afterwards fluctuation was recognised, and a surgical operation was decided upon. I may here say that I do not remember to have been impressed by any sense of wooden hardness or "board-like" resistance before the fluctuation, such as has been described by some authors as characteristic of the inflammations produced by actinomycosis.

It is well recognised that the only certain evidence of actinomycosis is the appearance of the pale yellow or greenish-yellow specks or granules in the secretion of pus from the affected part. These may be seen in the sputum of the pulmonary cases, and in the pus from lesions discharging on to the surface spontaneously or after incision; and they were found in the urine in the interesting case read by Dr. W. B. Ransom before the Royal Medical and Chirurgical Society in November, 1891.[1] But the granules may be overlooked, and in the present case nothing unusual was noticed in the pus on the day of the operation, or when the wound was subsequently dressed, and certainly no one recognised the granules of the ray fungus. Dr. W. H. Ransom, who also read an interesting case before the same Society,[2] recommends that in a suspected case the pus should be poured gently down the side of a glass, when granules can be readily recognised, and an examination under the microscope will at once determine the nature of the complaint. At the post-mortem examination of my own case the granules were seen as very minute specks, whiter than the yellowish-white pus in which they floated. But their colour seems to vary a good deal in different cases. Boström observes that they have been most often described as yellow, yellowish, sulphur-yellow, yellow-green, or yellow-brown; also as slightly yellow, pale yellow, bright yellow, grey yellow, dull yellow, light yellow, intensely yellow, dark yellow, citron-yellow, saffron-yellow, chrome-yellow; more rarely as white, whitish, or grey, dirty grey-white, grey-white, translucent grey, sago-like; and most rarely as green, greenish, grass-green, and dark green. Such differences probably depend on the age of the colony, and the relative proportions of mycelium threads to terminal clubs.

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[1] 'Brit. Med. Journal,' November 14th, 1891, vol. ii, p. 1046; 'Lancet' of same date.

[2] 'Brit. Med. Journal,' loc. cit., p. 1045.

The most common size of the free granules is between a quarter and two fifths of a millimetre, and the largest may reach to one or two millimetres, that is, $\frac{1}{25}$ or $\frac{1}{12}$ part of an inch.

If it is not clear how the fungus reached the interior of my patient, I think we can at least understand its progress when it once obtained a hold. It must have invaded the mucous membrane of the duodenum and the colon, and thence have reached the liver, probably by contiguity and spread of inflammatory lesions. In this organ appear the most extensive lesions, the largest of which, that in the anterior half of the right lobe (Fig. 1), was not confined to the limits of the organ, but spread to the adjacent tissues. From this, probably the first lesion, I suppose the other lesions in the liver (Figs. 1 and 2) as well as those in the lung must have occurred by particles of the fungus passing into the blood-current. Undoubtedly in a large proportion of cases the lesion is simply one of local irritation; and there appears to be no special condition of septic poisoning analogous to that in the specific fevers, diphtheria, or tetanus, apart from that which is referable to the suppuration induced by the presence of the fungus.

The disease in a number of cases extends in a remarkable manner from tissue to tissue, so that commencing in the intestines it may reach the liver and thence the abdominal parietes; or, perforating the œsophagus, it may lead to erosion of the spine, and beginning in the lung it will invade the pleura, and subsequently form an abscess under the skin. Boström denies that there is any metastasis to the lymphatic glands.

But in some cases at least the disease becomes spread more widely than can be accounted for by extension through contiguous adjacent structures. Secondary deposits have been observed in most of the organs, including the brain, and in the muscles and skin; and in the present case, if the large mass in the liver represented in Fig. 1 is the primary lesion, the white nodules and masses in the posterior and upper parts remote from the large mass, and the nodules in the lungs resembling tubercles can best, and perhaps only be explained by supposing they arise from particles of the fungus disseminated by "water carriage" through the viscera concerned. It may be said that the nodules in the lungs were due to primary infection by particles inhaled through the mouth, but when it is remembered that the trouble in the right flank commenced at the beginning of June, that his illness lasted in all three months, that he was in the hospital under observation for more than six weeks, that the lesions in the lungs were quite small and obviously recent, such a view has little in its favour. The only possibility of this kind would be that the fungus was present about the mouth or teeth for a long time, and in the last days or weeks of his illness some got inhaled into the lungs. It is true that there were two decayed molar teeth, but the jaws were entirely free from abscess, necrosis, or caries otherwise; and if these teeth were the source of the mischief in the lungs, they must have harboured the fungus in a quiescent condition for some time before it was inhaled.

The treatment consists in the removal of the affected tissues by surgical means where that is possible, as is sometimes the case where the disease involves only the neighbourhood of the lower jaw.[1] Where the viscera or deeper parts are involved, or very numerous centres of disease have formed, it is not clear that anything can be done to prevent a fatal issue, though Dr. W. B. Ransom's case appears to show that under certain circumstances the symptoms may disappear, and recovery take place.

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[1] See the first six cases by Boström, loc. cit., and Mr. Anderson's case, 'Brit. Med. Journ.,' 1891, vol. ii, p. 1046.

LIST OF SPECIMENS ADDED TO THE PATHOLOGICAL MUSEUM

DURING THE YEAR 1891.

By LAURISTON E. SHAW.

1083(43) Comminuted Fracture of the Skull causing Epilepsy.

Portions of a skull removed during life. The two larger pieces were removed by the trephine, after which the smaller fragments were found beneath the dura mater, embedded in the substance of the brain. One of the trephine circles shows a small oval perforation with irregular edges.

Ernest W., æt. 20, was admitted under Mr. Davies-Colley, in 1890, with a sinus on the right side of the scalp, at the bottom of which bare bone was felt. The sinus, from which portions of necrosed bone had been twice removed, followed an injury received two years before admission in being thrown from a horse. The patient had two fits after a head injury at the age of ten years, since which time there had been no recurrence of fits until within the eight months preceding his admission to the hospital. During this period he had had ten fits, which began in, and chiefly affected, the left side of his body. He was discharged well three weeks after the operation. *See Surgical Reports*, vol. cxlvii, Case 97.

1107(23) Secondary Epithelioma of the Humerus.

The upper two thirds of a left humerus divided longitudinally, showing a portion of its shaft four inches from its upper extremity, replaced by an exceedingly soft white growth.

The upper and lower parts of the bone thus spontaneously fractured are separated by an interval of two inches. The growth displaces and in parts invades the adjacent muscles, extends into the medullary canal of the bone, and for a short distance separates the periosteum from the shaft. Histologically the growth is a squamous-celled epithelioma.

George P., æt. 40, was admitted under Mr. Lane in 1890 for a spontaneous fracture of the left humerus, which had taken place a few weeks previously while the patient was under treatment for symptoms of intra-thoracic growth. The arm was amputated at the shoulder-joint, and the patient made a good recovery from the operation. *See Surgical Reports*, vol. cl, Case 44.

1158(56) **Stump after Stephen Smith's Amputation.**

An antero-posterior section through a stump resulting from the operation known as Stephen Smith's lateral flap amputation at the knee. There is seen to be an inch and a half of soft tissues covering the end of the femur, and the patella is drawn up upon the anterior surface of the bone. The section through the stump has divided two small deposits of melanotic sarcoma in the skin.

Sarah W., æt. 55, was admitted under Mr. Lucas in 1890 for a recurrence of melanotic sarcoma, for which a circular amputation through the middle of the thigh was performed. Three and a half years previously Mr. Bryant had removed the leg by Stephen Smith's amputation. The patient made a good recovery after each operation. *See Surgical Reports*, vol. cxlviii, Case 85.

1361(51) **Subcutaneous and Intra-muscular Abscesses from Glanders.**

A portion of skin and subjacent tissues from a thigh, showing two white nodules about an inch in diameter, one of which is situated immediately beneath the skin, while the other lies more deeply in the muscle. In the recent state these nodules were found to consist of gelatinous pus, which contained the specific micro-organisms of glanders.

Henry A., æt. 53, an engineer, was admitted under Dr. Pavy for pains in the limbs, fever, and numerous superficial abscesses. He died six days after the onset of acute symptoms, and at the autopsy abscesses were found in his lungs. *See Insp.*, 1889, No. 408; and *Guy's Hosp. Reps.*, 1890, vol. xlvii, p. 138.

1366(60) Sarcoma of the Tendo Achillis.

A vertical section through the lower third of a left leg and foot, showing a firm oval tumour occupying the position of the tendo Achillis. The growth extends from the os calcis upwards for about four inches. Its cut surface is in parts smooth and fibrous, in others rough and gritty from calcareous deposit. On the reverse of the specimen the skin covering it is seen to be ulcerated. Histologically the growth is a sarcoma with large oval and round cells containing masses of cartilage scattered through it.

Henrietta B., æt. 17, was admitted under Mr. Lucas in 1890, for a painful swelling behind the left heel, which had been noticed for six months and had been rapidly enlarging for two months. Three years before admission there had been some swelling of the ankle, attributed to an injury. The leg was amputated, and the patient was discharged well. *See Surgical Reports*, vol. cxlviii, case 89.

1504(36) Arterio-venous Aneurysm of the Splenic Vessels.

A spleen, with a portion of the stomach and the splenic artery and vein. Both the splenic vessels are much dilated, the artery being also very tortuous. At the hilus of the spleen there is a saccular dilatation of the vein as large as a hen's egg, containing adherent thrombus, and communicating by a rounded perforation with a similar, but much smaller, dilatation of the artery. On the reverse of the specimen the coronary veins of the stomach and the branches of the splenic vein in the substance of the spleen are seen to be filled with ante-mortem blood-clot. The spleen is enlarged and has recent lymph upon its capsule.

Matilda J., æt. 49, was admitted under Dr. Goodhart for ascites with tenderness in the region of the spleen, over which area a loud, "humming," systolic murmur was heard. She had suffered from vomiting, with occasional hæmatemesis for three years. Blood-stained fluid was removed from the peritoneum by paracentesis. She had muscular twitchings and became comatose, and died one month after admission. At the autopsy several decolorised infarcts were found in the spleen, and the splenic and superior mesenteric veins contained adherent thrombus. The colon was congested, the heart was normal, the kidneys were granular. *See Insp.*, 1888, No. 226; and *Trans. Path. Soc.*, vol. xl, p. 67.

1504(46*a*) **Ruptured Aneurysm of the Superior Mesenteric Artery.**

A portion of an abdominal aorta showing a saccular aneurysm attached to the posterior surface of its superior mesenteric branch. The aneurysm arises one inch from the aorta and extends along the artery for two inches. It is oval in shape, with the largest diameter, which measures four inches, placed transversely. On the reverse of the specimen, the sac presents a ragged perforation through which its interior may be seen to be partly filled with laminated blood-clot.

E. B., a man æt. 54, was admitted to the St. George's Infirmary for sudden abdominal pain and collapse. A pulsating tumour was felt in the abdomen. He died thirty-six hours from the onset of his illness. At the autopsy a large amount of blood was found effused into the retro-peritoneal tissues, and the peritoneal cavity contained two pints of blood-stained fluid. *See Note Book*, 3, p. 12.

Presented by Mr. C. POLLARD.

1519(70) **Symmetrical Aneurysms of the Vertebral Arteries.**

A cerebellum, with the pons varolii and medulla oblongata, showing two small aneurysms situated symmetrically, one upon each vertebral artery, about half an inch from the commencement of the basilar artery. The aneurysm upon the left side, which is the smaller, shows a rent in its wall; that upon the right side is three eighths of an inch in diameter and is filled with ante-mortem thrombus.

George G., æt. 25, was admitted under Dr. Hale White for headache, vomiting, and optic neuritis. The left side of the face and the right side of the tongue were paralysed. Soon after admission the left side of the tongue also became paralysed, and he died suddenly about five weeks from the commencement of his illness. At the autopsy the mitral valve was contracted, and upon its anterior cusp there were recent vegetations, in which micrococci were found. There were infarcts in the spleen and kidneys and a considerable effusion of blood at the base of the brain in the subarachnoid space. *See Insp.*, 1889, No. 365; and *Trans. Path. Soc.*, 1890, p. 60.

1562(66) **Tuberculous Spinal Pachymeningitis.**

The lower end of a spinal cord, with a portion of the cauda equina, divided by a longitudinal incision to show a thick deposit of caseous material attached to the inner surface of the dura mater, and forming a complete sheath around the cord. Histologically this deposit consists of small round cells, with numerous foci of caseation and a few giant cells. On the reverse of the specimen a small sequestrum from a lumbar vertebra may be seen adherent to the anterior surface of the dura mater.

From a boy. æt. 4½, who died from tuberculous cerebral meningitis. He had suffered for a long time from caries of the spine with an iliac abscess. Before his death there was marked weakness of the lower extremities. *See Trans. Path. Soc.*, vol. xli, p. 23.

Presented by Mr. L. A. DUNN, 1890.

1567 Cerebral Hæmorrhage in Purpura.

A coronal section through a brain at the level of the optic commissure, seen from behind. In the left lenticular nucleus there is a rounded extravasation of blood half an inch in diameter. In the recent state the surrounding brain substance was soft and of a yellowish colour.

Martha H., æt. 28, was admitted under Dr. Goodhart for purpura hæmorrhagica of three weeks' duration. There was hæmorrhage from the gums and nose and into the retinæ. She gradually sank into a comatose condition, and died two months after her admission. At the autopsy numerous petechiæ were found throughout the body, and there were small extravasations of blood upon the surface of the brain. *See Insp.*, 1889, No. 384.

1567(5) Cerebral Hæmorrhage extending into the Lateral Ventricles.

A horizontal section through a brain, laying open the lateral ventricles, which are seen to be distended with blood-clot. In the right hemisphere the brain substance outside the ventricle is occupied by a large extravasation of blood, of which the hæmorrhage into the ventricles is an extension.

Maria P., æt. 67, was admitted under Dr. Pavy in a comatose condition, with unequal pupils and albuminuria, and died two and a half hours from the onset of her illness. While in fair health she suddenly became giddy and aphasic, and lost the power of her left leg. An hour later she was quite unconscious. At the autopsy the heart was found to be hypertrophied; the kidneys were scarred and granular. *See Insp.*, 1888, No. 366.

1575(12) **Hæmorrhagic Cyst of the Brain.**

A horizontal section through the left cerebral hemisphere showing an irregular excavation about an inch in its longest diameter, situated behind the lenticular nucleus and destroying the posterior part of the internal capsule. Its lining membrane is smooth, and in the recent state the cyst contained a light-brown fluid.

Benjamin B., æt. 62, was admitted under Mr. Durham for an aneurysm of the axillary artery, from which he died. Three years before his death he had a sudden attack of right hemiplegia with slight aphasia. When attacked he fell down. He gradually recovered, but a feeling of numbness in the left arm persisted for many weeks. At the autopsy the heart was found to be hypertrophied and the vessels of the brain were atheromatous. The kidneys were healthy. No infarcts were found. *See Insp.*, 1888, No. 396.

1587(25) **Tuberculous Inflammation of the Dura Mater invading the Brain.**

A portion of the right half of a brain showing the dura mater firmly adherent to the frontal and temporo-sphenoidal lobes on either side of the sylvian fissure. At the situation of the adhesion the external surface of the dura mater is roughened by inflammatory material, and the section which has been made through it shows that the brain substance is invaded to a depth of half an inch by firm caseous deposit.

Geo. R., æt. 10, was admitted under Dr. Goodhart for a caseous abscess in the right temporal region, which had been noticed for fourteen days. The abscess was opened and portions of the frontal and temporal bones were felt to be bare and rough. He died six months from the onset of his illness from miliary tuberculosis of the lungs and pleurisy. At the autopsy the skull was found to be perforated just behind the right external angular process of the frontal bone. *See Insp.*, 1890, No. 98; and *Prep.*, 3401.

1591(80) **Subdural Hæmorrhagic Cyst.**

A portion of dura mater from the left side towards the frontal region, showing loosely attached to its cerebral surface a thin-walled cyst. The long axis of the cyst has an antero-posterior direction, and both ends have been opened in taking the part from the body; its transverse diameter is two inches. In the recent state it contained a thin, light-red fluid and a small quantity of blood-clot.

From a man, æt. 46, who had suffered for two years from severe headache, and during the month preceding his death had twitchings of the arms and face. There was no paralysis nor anæsthesia; his urine was free from albumen. He became comatose one week before his death. At the autopsy a smaller cyst was found symmetrically placed beneath the dura mater on the right side, the surface of the brain being markedly compressed. *See Note Book*, No. 3, p. 11; and *Trans. Path. Soc.*, vol. xliii.

Presented by Mr. G. H. Knapp, 1890.

1592(51) Tuberculous Tumour of the Dura Mater.

The posterior part of the dura mater with the falx and tentorium cerebelli. Attached to the entire upper surface of the tentorium on the left side there is a nodulated mass of growth about two inches in diameter, which in the recent state compressed the occipital lobe. Histologically the growth has the structure of caseating tubercle and contains numerous giant-cells.

From a woman, æt. 20, who, after a short period of delirium, became comatose and died. She was said to have enjoyed good health until the onset of her fatal illness.

Presented by Dr. Cameron Brock, 1889.

1602(35) Sarcoma of the Dura Mater invading the Brain.

An oval nodular mass of growth removed during life from the frontal region of the right side of a brain. It measures four inches long, three inches broad, and two and a half inches in thickness, and in the recent state weighed six and a half ounces. The growth, which is covered for the most part by a delicate membranous capsule, is firm and its cut surface presents a white fibrous appearance. Histologically it has the structure of spindle-celled sarcoma. The two trephined circles of bone mounted below show the extreme thinning of the skull produced by the pressure of the tumour.

Henry A., æt. 31, was admitted under Dr. Goodhart for headache and vomiting, with weakness and rigidity of the left arm and leg. These symptoms had been gradually increasing for two years. Mr. Symonds removed the growth and the patient died seven days later from facial erysipelas. At the autopsy it was found that a portion only of the growth

had been removed. The remainder was attached to the falx cerebri, and from this it appeared that the tumour had originally sprung. There was no meningitis. *See Insp.*, 1890, No. 434.

1652(46) Partial Calcification of a Lipoma.

Half of a large fatty tumour, the cut surface of which shows that a considerable portion of it has undergone calcareous degeneration.

The tumour was removed from the deltoid region of a patient who had noticed its existence for forty years. The growth had increased in size rapidly previous to its removal.

Presented by Mr. LAWFORD KNAGGS, 1890.

1657(11) Mycosis Fungoides.

A portion of skin from the back showing a flattened tumour about seven inches long by six inches wide, which at its most prominent part projects two and a half inches from the surrounding surface. The growth is superficially ulcerated, and in parts gangrenous. Histologically the growth appears to be due to a diffuse infiltration of the corium and subcutaneous tissues with small round cells.

John B., æt. 66, was admitted under Dr. Pye-Smith for general dermatitis and multiple tumours of the skin, some of which sloughed. He died from gangrenous broncho-pneumonia, and at the autopsy the right supra-renal capsule was infiltrated by a growth similar in character to those in the skin. *See Insp.*, 1891, No. 107; *Prep.*, No. 2022^{14}; *Clin. Soc. Trans.*, vol. xxv; and drawings.

1799(37) Sloughing of Gastric Mucous Membrane in Corrosive Poisoning.

A stomach seen from behind showing upon its anterior wall towards the pyloric end a yellow slough which consists of mucous membrane separated from the muscular coat of the organ. There is a smaller slough at the cardiac end of the stomach and the intervening mucous membrane presents several deep ulcers.

Anne P., æt. 18, was admitted under Dr. White, having swallowed some chloride of zinc dissolved in hydrochloric acid. She died on the eleventh day after the accident, having suffered from hæmatemesis and melæna on the day before her death. At the autopsy the cæcum was much congested. *See Insp.*, 1887, No. 287.

1799(38) Poisoning by Hydrochloric Acid.

Portions of a stomach and œsophagus mounted to show their coats blackened by the action of hydrochloric acid.

Presented by Dr. BIRT, 1889.

1801(11) A Perforation in a Varicose Vein of the Stomach.

A portion of the cardiac end of a stomach showing upon its anterior wall, two inches from the œsophageal opening, a minute circular aperture with thin membranous edges. On the reverse of the specimen may be seen a dilated tortuous branch of the gastric vein, its communication with the opening on the mucous surface being indicated by a red rod.

John E., æt. 17, was admitted under Dr. Taylor for hæmatemesis, and died four days after admission from continual loss of blood. At the autopsy the liver was found to be cirrhosed and contracted. Water injected into the coronary vein of the stomach flowed freely from the opening in the mucous membrane. *See Insp.*, 1889, No. 211.

1802(6) Post-mortem Solution of the Stomach.

A portion of the anterior wall of a stomach, showing upon its mucous membrane numerous excavations, oval or round in shape, and varying in size from one to five millimetres in diameter. Towards the greater curvature these excavations are so closely aggregated as to produce a worm-eaten appearance of the lining of the stomach. Their edges are for the most part abrupt and the deeper ones expose the muscular coat. The intervening mucous membrane appears healthy, and histologically shows no sign of inflammatory action.

James H., æt. 42, was admitted under Dr. Taylor with an empyema following lobar pneumonia. The thorax was incised and the patient died three days after the operation. *See Insp.*, 1891, No. 116.

1802(76) Perforating Ulcer of the Stomach.

The cardiac half of a stomach, showing two ulcers situated half-way between the œsophageal and pyloric orifices, one upon the anterior, the other upon the posterior wall of the organ. The edges of the former are somewhat thickened and sharply defined, and the opening through the peritoneal coat is sealed by a firm layer of recent lymph. The serous coat, which is exposed in the base of the other ulcer, is extremely thin.

Christina F., æt. 19, was admitted under Dr. Shaw for abdominal pain and collapse, for which an exploratory laparotomy was performed. She had suffered from dyspepsia and vomiting after food for eighteen months. Her death took place five days from the onset of urgent symptoms. At the autopsy general peritonitis was found. *See Insp.*, 1890, No. 352.

1805(21) Ulcers of the Stomach and Pylorus.

The first part of a duodenum with the adjacent portion of the stomach showing two deep ulcers situated upon the pyloric ring and extending into the intestine. On the pylorus the ulceration is superficial, while in the intestine the process has extended more deeply and in the base of the larger ulcer exposes the serous coat. There are two small round ulcers upon the lesser curvature of the stomach, about an inch and a half from the pylorus.

William W., æt. 39, was admitted under Mr. Davies-Colley with cellulitis of the neck, and died fourteen days from the onset of his illness. At the autopsy the thyroid cartilage was found to be necrosed, and there was a cellular abscess in the lumbar region. *See Insp.*, 1891, No. 118.

1806(76) Traumatic Stenosis of Pylorus.

A stomach and a part of the duodenum, showing considerable thickening of the pyloric ring with narrowing of its orifice so that in the recent state it barely admitted a No. 4 catheter. The stomach is small and its walls are thickened, especially towards the pyloric end. There is no ulceration nor scarring of its inner surface. Half an inch from the pyloric ring and parallel to it there is upon the anterior surface of the organ an incision about an inch long, around the margin of which there are the marks of sutures and a little granular lymph.

Henry G., æt. 43, was admitted under Dr. Hale White for vomiting and dilatation of the stomach. Seven weeks before admission he drank some "spirits of salts" and suffered for three days from symptoms of corrosive poisoning. A month later vomiting came on which was unassociated with pain and gradually increased in frequency. On admission he was extremely emaciated. Mr. Lane opened the stomach and passed a catheter into the duodenum through which the patient was fed. He died on the sixth day after the operation. At the autopsy some of the sutures uniting the stomach to the abdominal wall were found to be loose, the result, it was thought, of post-mortem solution, and there was food in the peritoneal cavity. There was no general peritonitis. *See Insp.*, 1890, No. 381; and *Trans. Clin. Soc.*, vol. xxiv, p. 108.

1911(60) Fatty and Cirrhotic Liver.

A portion of liver the cut surface of which shows many of the larger branches of the hepatic vein to be surrounded by well-defined zones of a pale yellow colour. These pale areas are seen under the microscope to be produced by extreme fatty infiltration of the liver-cells, whilst the rest of the organ, which presents no abnormal appearance to the naked eye, shows an excess of fibroid tissue. The surface of the liver is smooth.

Thomas H., æt. 42, was admitted under Dr. Goodhart for enlargement of the liver and spleen with jaundice and pyrexia. There was pus in the urine. He died from facial erysipelas. At the autopsy pyelonephritis was found. *See Insp.*, 1890, No. 414; and Drawing.

2020(7) Adenoma of the Supra-renal Capsule.

A right supra-renal capsule from the upper surface of which projects a smooth ovoid tumour two and a quarter inches in its longest diameter. The tumour is covered by the delicate fibrous investment of the supra-renal body, and its cut surface presents the appearance of the cortical substance of the organ. Histologically the growth has the structure of the supra-renal capsule infiltrated with oil globules.

Mary D., æt. 57, was admitted under Mr. Davies-Colley for carcinoma of the uterus. At the autopsy there was found to be a hydronephrosis on the left side. The left supra-renal was normal. *See Insp.*, 1889, No. 141.

2022(14) Supra-renal Capsule in Mycosis Fungoides.

A right supra-renal capsule enlarged so as to weigh four and a half ounces. It is infiltrated and replaced by a soft growth, which in the recent state was vascular and in parts yellow and caseous. Histologically the growth consists of an aggregation of small round-cells with numerous areæ of necrosis.

John B., æt. 66, was admitted under Dr. Pye-Smith for general dermatitis and multiple tumours of the skin, some of which sloughed. He died from gangrenous broncho-pneumonia. *See Insp.*, 1891, No. 107; *Prep.* 1657^{11}; *Clin. Soc. Trans.*, vol. xxv; and drawings.

2071(52) Hydronephrosis. Compression of Ureter by Renal Vein.

A left kidney, the pelvis and calices of which are much dilated. At its junction with the pelvis the ureter is crossed and compressed by a large branch of the renal vein. In the recent state the pelvis was distended by urinous fluid which could not be forced into the ureter until the relation of the parts was disturbed.

Sarah N., æt. 36, was admitted under Dr. Pye-Smith for epileptiform fits, which continued till her death twelve hours after admission. At the autopsy the right kidney was found to be hypertrophied. *See Insp.*, 1889, No. 424.

2228(94) Parovarian Cyst.

A spheroidal cyst about four and a half inches in diameter, which was removed during life from the left broad ligament. The cyst is unilocular, and its lining is smooth and fibrous. The left fallopian tube and ovary are attached to its outer surface.

From M. L., a middle-aged married woman, who had noticed the presence of an abdominal tumour for about twelve months. At the operation the cyst was found to be firmly attached to the side of the uterus. It contained blood-stained fluid. The patient made a good recovery.

Presented by Dr. Galabin, 1890.

2253 Hydrosalpinx.

A right fallopian tube, the distal portion of which is greatly dilated so as to form a kidney-shaped cyst, measuring five inches in length and three inches at its broadest part. The wall of the cyst is thick and fibrous, and in the recent state it contained eight ounces of clear fluid.

Sarah C., æt. 40, was admitted under Dr. Galabin for a tumour above Poupart's ligament on the right side, which had been noticed for ten months. Laparotomy was performed, and both fallopian tubes were removed. The left, which was much less distended than the right, contained pus. The patient was discharged well one month after the operation. *See Obstetric Reports*, 1891.

2254(46) Pyosalpinx.

A much thickened and convoluted left Fallopian tube, the distal end of which is dilated so as to form an ovoid sac measuring four inches in its longest diameter. The fibrous wall of the sac is in some places three eighths of an inch thick, and its peritoneal surface presents the remains of adhesions to neighbouring structures. In the recent state it contained thick pus.

Emma S., æt. 43, was admitted under Dr. Galabin, who removed the Fallopian tube by laparotomy. *See Obstetrical Reports*, 1890.

2302(1) Scirrhous Carcinoma of the Breast.

A section through the centre of a breast showing the gland tissue beneath the nipple to be invaded by a white fibrous growth.. The nipple is depressed. Histologically the growth is a spheroidal carcinoma having a large amount of stroma.

From Miss R., æt. 21, who had noticed the existence of the tumour for six months. There was no family history of cancer, and no injury to the part had been sustained. The breast was removed, but the growth recurred, and the patient died from secondary deposits in the lung fourteen months after the operation.

Presented by Mr. BRYANT, 1889.

2365(70) Cystic Epididymis.

A testis, the epididymis of which is greatly enlarged, and contains numerous cysts with thin translucent walls. One of the cysts measures two inches in diameter, and the whole epididymis is more than four times as large as the testis. The cysts have a smooth lining, and in the recent state were filled with an opalescent fluid containing numerous spermatozoa.

Samuel W., æt. 65, was admitted under Dr. Goodhart for chronic Bright's disease of ten years' duration, and died from uræmia. The other epididymis contained some cysts, and histological examination of its more solid part showed some of the tubules to be slightly dilated. *See Insp.*, 1890, No. 57.

2367 (81) Melanotic Sarcoma of the Spermatic Cord.

A left testicle showing in the spermatic cord about an inch above the globus major a flattened oval mass of melanotic growth, which measures two and a half inches in its longest diameter. Its upper part is soft and diffluent, while the lower portion is more solid, and presents one or two white nodules in its substance. Histologically the growth is a spindle-celled sarcoma, and contains much black pigment.

Charles D., æt. 70, was admitted under Mr. Lane in 1891 for a painless enlargement in the left groin which had been noticed for six months. He had worn an inguinal truss for nine years. Three years before admission his left eye was excised at the London Hospital. The parts shown in the preparation were removed together with an enlarged iliac gland, and the patient recovered from the operation. *See Surgical Reports*, 1891.

2507 (1) Richter's Hernia.

A piece of small intestine excised during life. It is laid open along its mesenteric attachment to show a hemispherical sacculus about the size of a walnut. The walls of the sacculus are thin and lined by mucous membrane which shows superficial ulceration. At the operation the protrusion was found in the sac of a femoral hernia.

Elizabeth C., æt. 53, was admitted under Mr. Lane in 1890 for a strangulated femoral hernia. Herniotomy was performed, a loop of intestine including the strangulated portion was excised, the ends being united by means of Senn's plates. The patient recovered. *See Surgical Reports*, 1891; and *Trans. Clin. Soc.*, vol. xxiv, p. 182.

***A3110a Adenomata of the Thyroid Gland.**

A thyroid gland seen from behind showing the left lobe to be almost entirely occupied by a globular encapsuled tumour about an inch and a half in diameter. Its cut surface presents numerous small cysts containing colloid material embedded in a spongy and vascular matrix. Histologically the adenoma has a basis of fibro-hyaline tissue, in which are numerous vesicles of various sizes, some of which are filled with epithelial cells, while others have a lining of cubical epithelium, and contain colloid material. At the apex of the right lobe there is a second encapsuled adenoma half an inch in diameter, consisting of a homogeneous soft material, which in the recent state was pink and vascular. Histologically this adenoma differs from the adult thyroid in the smaller amount of fibrous tissue and the greater proportion of colloid material.

Mary B., æt. 49, was admitted under Dr. Horrocks for inversion of the uterus of long standing, and died the day after the operation of vaginal hysterectomy. *See Insp.*, 1890, No. 265.

3172a Trachea eroded by Aneurysm.

The arch of an aorta with the lower end of the trachea. There is a saccular aneurysm, about the size of a Tangerine orange, situated between the origins of the innominate and the left common carotid arteries, in the posterior wall of which are exposed portions of seven tracheal rings, some of them being eroded. On the reverse of the specimen the mucous membrane of the trachea presents just above its bifurcation an opening, through which protrudes a small pea-shaped nodule of firm decolorised blood-clot.

Michael C., æt. 48, was admitted under Dr. Perry for aphonia and dyspnœa. His dyspnœa became urgent, and he died from venous hæmorrhage while tracheotomy was being performed for its relief. *See Insp.*, 1890, No. 357.

3237 Bronchus invaded by Sarcoma of the Mediastinal Glands.

The lower part of a trachea with the right lung attached. The upper division of the right bronchus, indicated by a blue rod, is surrounded and much constricted by a soft white growth which is continuous with a mass at the root of the lung. Almost the whole of the lower lobe is occupied by a cavity with rough trabeculated walls surrounded by pneumonic lung. There are similar smaller cavities in the middle lobe and the lower part of the upper lobe, and numerous small excavations elsewhere in the pulmonary tissue. On the reverse of the specimen the mediastinal growth may be seen to surround and invade the superior vena cava. The pleura is greatly thickened. Histologically the tumour is a small round-celled sarcoma.

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* Numbers in this Appendix above 3000 refer to preparations added to that part of the Museum of which the new Catalogue is already written.

Ellen S., æt. 38, was admitted under Dr. Taylor with physical signs indicating destruction of the right lung. She died a few hours after admission. At the autopsy nodules of growth were found in the mesenteric glands, ovary, and pancreas. The left lung was quite healthy. *See Insp.*, 1885, No. 306.

3239 Bronchus compressed by Mediastinal Sarcoma.

The lower part of a trachea with the main divisions of the bronchi. The left bronchus is invaded and compressed by a soft white growth af the root of the lung. Histologically the growth is a round-celled sarcoma.

Charles K., æt. 42, was admitted under Dr. Goodhart for effusion into the left chest, which was found upon aspiration to be blood-stained. A month later twenty-four ounces of pus were removed from the chest. He died fifteen weeks from the commencement of his illness. At the autopsy there were secondary deposits of sarcoma in the left pleura, and the lung was in a condition of disintegrating broncho-pneumonia. *See Insp.*, 1890, No. 304.

3252 Traumatic Hæmorrhage into the Lung.

The lower lobe of a left lung showing a blood-clot two inches in diameter embedded in the pulmonary tissue towards the base. On the reverse of the specimen the pleura is seen to be lacerated in several places.

Henry B., æt. 40, was admitted under Mr. Durham, having been struck on the left side by a lever. There was some hæmoptysis. He died one week after admission. At the autopsy the left pleural cavity contained much blood, and five ribs on that side were fractured and had lacerated the parietal pleura. The spleen was also torn. *See Insp.*, 1890, No. 266.

3261a Brown Induration of the Lung.

A portion of the lower lobe of a lung, the cut surface of which shows the pulmonary tissue to be unusually firm, and in parts consolidated. In the recent state the consolidated portions were brown in colour. There is flocculent lymph upon the pleura. Histologically the alveolar walls are thickened, and many of the spaces are filled with red blood-corpuscles.

Alfred W., æt. 12 years, was admitted under Dr. Pavy for mitral stenosis. He had an attack of acute rheumatism six years previously. During the last month of his life he suffered from hæmoptysis and occasional pyrexia. At the autopsy both lungs were partly consolidated, and in the upper lobe of the right lung there was a localised extravasation of blood. *See Insp.*, 1890, No. 358.

3273 Broncho-pneumonia with Cavitation and Bronchiectasis.

Portions of lung showing scattered patches of broncho-pneumonia and numerous minute cavities. Some of these are formed by dilatations of the bronchial tubes, and others are excavations in the pneumonic lung. There is granular lymph upon the pleura. Histologically there is no evidence of tubercle.

Ernest E., æt. 2, was admitted under Dr. Pye-Smith for broncho-pneumonia, and died two days after admission. *See Insp.*, 1890, No. 142.

A3284 Actinomycosis of the Lung.

A section of the lower lobe of a lung, the cut surface of which presents several nodules of consolidated lung tissue about half an inch in diameter. In the recent state these nodules consisted of a soft central white area surrounded by a deep red zone. Under the microscope the centre of the nodule is seen to be made up of small round cells, among which are numerous actinomyces, while in the periphery the alveoli of the lung are filled with extravasated red blood-corpuscles.

Henry L., an engraver, æt. 42, was admitted under Dr. Taylor for an abscess in the lumbar region, which was incised and drained. He was much emaciated, and had been ill for three months before admission. The discharge from the abscess almost ceased and the cavity contracted, but the temperature remained high, and the patient becoming gradually exhausted, died during an attack of syncope six weeks after the operation. At the autopsy the lumbar abscess was found to communicate with a ragged cavity at the lower part of the right lobe of the liver, in the walls of which were found numerous ray fungi. Several smaller actinomycotic nodules were found in the rest of the liver and throughout both lungs. *See Insp.*, 1890, No. 330.

3284 Diffuse Syphilitic Fibrosis of the Lung.

A portion of a right lung, in which there is a considerable excess of fibroid material appearing on the cut surface as a delicate network traversing the pulmonary tissue in all directions. This fibroid change is less marked at the apex than at the base; in the latter situation the air-vesicles are many of them dilated. The pleura is slightly thickened, and was adherent. Histologically the dense fibroid tissue that pervades the lung shows scattered through it numerous collections of small round cells not undergoing caseation. No giant cells are seen. The walls of the small arteries are thickened.

John A., æt. 54, was admitted under Dr. Taylor with severe bronchitis and dyspnœa of one week's duration. He had suffered from winter cough for some years. At the autopsy the testes were fibroid, the liver was scarred, and the kidneys were lardaceous. The left lung was affected in the same way as the right. *See Insp.*, 1889, No. 451.

3297 Miliary Tuberculosis with Cavitation of the Lung.

A portion of the lower lobe of a right lung, the cut surface of which is studded with miliary tubercles. At the lower and hinder part of the lobe there are numerous small excavations with ragged walls, which in the recent state contained pus. There is granular lymph upon the pleura. Histologically there are patches of tuberculous broncho-pneumonia with central disintegration.

Henry S., æt. 5, was admitted under Dr. Shaw with general tuberculosis and physical signs of broncho-pneumonia. The duration of his illness was seven weeks. At the autopsy tubercles were found in the kidneys, spleen, and liver, and some of the branches of the pulmonary veins contained adherent thrombus. *See Insp.*, 1890, No. 305.

3349a Epithelioma of the Lung with Cavitation.

A left lung, the lower two thirds of which is hollowed out, forming a cavity measuring four and a half inches in its vertical and antero-posterior diameters. The thin layer of pulmonary tissue and pleura which forms the wall on its lateral aspect has been partially removed, and shows the opening of the main bronchus into the cavity, which is indicated by a blue rod. In the recent state the interior was shaggy and covered with muco-purulent secretion. On the reverse of the specimen a section through the consolidated base of the lung displays sinuous cavities resulting from dilatation of bronchial tubes. The pleura over the lower lobe is much thickened and was adherent. Histological examination of the wall of the cavity shows the pulmonary tissue to be infiltrated by a malignant growth having the characters of a squamous-celled epithelioma.

William A., æt. 68, was an out-patient for some months, suffering from profuse blood-stained muco-purulent expectoration, the quantity of secretion sometimes exceeding a pint in the twenty-four hours. This condition continued for twenty months, towards the end of which time the patient became affected with anasarca. He died from cardiac failure. The post-mortem examination was incomplete.

Presented by Dr. Newton Pitt, 1889.

A3351 Scirrhous Carcinoma of the Lung.

A portion of a lung showing upon its surface numerous puckered depressions caused by the contraction of secondary deposits of growth beneath the pleura. Upon the cut surface of the lung may be seen two nodules of growth, one towards the lower part of the preparation situated immediately beneath one of the depressions, the other at the apex embedded deeply in the pulmonary tissue. Histologically the growth is a spheroidal-celled carcinoma with much fibroid stroma.

Maria O., æt. 50, was admitted under Dr. Goodhart with a hard tumour of the breast, jaundice, and enlarged liver. Retraction of the nipple had been noticed for eight or nine years. At the autopsy the tumour of the breast was found to be a scirrhous carcinoma, and there were secondary deposits in the liver. *See Insp.*, 1891, No. 18.

3401 Tuberculous Pleurisy.

A section through a left lung showing the visceral layer of the pleura and the interlobar septum universally thickened, in some places to the extent of one third of an inch. The surface presents delicate membranous adhesions, and in contact with it at the apex of the lung there are some caseous lymphatic glands. The pulmonary tissue contains miliary tubercles. Histologically the thickened pleura shows many caseous foci crowded with the bacilli of tuberculosis.

George R., æt. 10, was admitted under Dr. Goodhart for an abscess in the temporal region associated with caseous disease of the bone. Symptoms of bronchitis and pleurisy developed soon after admission, and the patient died six months from the first onset of his illness. At the autopsy there was found extension of the caseous disease of the temporal bone to the subjacent dura mater with general tuberculosis of the viscera. The right pleura was unaffected. *See Insp.*, 1890, No. 95; and Prep. 1587(25).

3404 Chronic Empyema with Deformity of the Chest.

The greater portion of the left side of a thorax, showing a lateral curvature of the spine with the convexity of the curve towards the right side. The ribs are crowded together, and the side of the chest is much flattened. The pleural cavity is lined with membranous lymph, and in the recent state contained pus. There is an opening in the sixth intercostal space just external to the cartilages of the ribs, indicated by a blue rod. The lung is compressed, and there is granular lymph upon the pericardium.

Florence M., æt. 15, was admitted under Dr. Goodhart for empyema, which had spontaneously opened, and was discharging pus from a sinus in the sixth left intercostal space. The sinus was enlarged and a drainage-tube inserted; the patient died one month after admission. At the autopsy there was pericarditis, but no lardaceous disease or tuberculosis. *See Insp.*, 1886, No. 187.

3445 Sarcoma of the Mediastinum.

A median vertical section through a sternum and the subjacent structures, showing the anterior mediastinum to be occupied by a firm white growth lying in contact with the posterior surface of the bone throughout its length, and displacing backwards the trachea and pericardium. The innominate artery and the left innominate vein are surrounded by growth, the vein being almost obliterated. The growth has invaded and penetrated the upper part of the gladiolus, and the muscles of one of the neighbouring intercostal spaces. On the reverse of the specimen the left pleura is seen to be covered by masses of growth, and in the recent state the pleural cavity contained about a pint of serous effusion. The lung is compressed, and its surface partially covered by a thin layer of granular lymph. There are secondary deposits in the enlarged cervical glands. Histologically the tumour is a small round-celled sarcoma.

Augusta J., æt. 25, was admitted under Dr. Goodhart for enlarged glands in the neck, and symptoms of mediastinal tumour. The glands were first noticed three months before admission. She died three weeks later, and during this time paracentesis thoracis was three times performed. At the autopsy the viscera were hard and congested, and the right pleural cavity contained two and a half pints of serous effusion. *See Insp.*, 1890, No. 244.

3448 Dermoid Cyst of the Mediastinum.

A heart showing an ovoid cyst attached to the external surface of the pericardium over the situation of the right auricle. The cyst, which is of about the size of a duck's egg, was slightly adherent to the right lung. It has a thin fibrous wall lined with squamous epithelium, to which in some places are attached long brown hairs. In the recent state it was filled with sebaceous material and a turbid fluid containing oil-globules and plates of cholesterine.

Maria J., æt. 20, was admitted under Dr. Hale White for meningitis of a fortnight's duration. She died the next day, and at the autopsy basal meningitis was found. *See Insp.*, 1890, 126; and *Trans. Path. Soc.*, vol. xli, p. 283.

3454 Epithelioma of the Lip with Secondary Deposits.

The left angle of the mouth with a portion of the cheek, showing upon the mucous surface an irregular oval ulcer about one and a half inches in its longest diameter, having a nodular base and thick raised edges. Below is mounted the left submaxillary salivary gland, to which is attached a lymphatic gland much enlarged and presenting a central excavation from the softening of secondary deposit. Histological examination of the ulcer and the enlarged gland show that the growth is a squamous-celled epithelioma.

Cornelius P., æt. 48, was admitted under Dr. Davies-Colley in 1890 for an ulcer on the buccal surface of the left cheek, and a swelling in the submaxillary region. The ulcer began as a blister two years before admission, and the glandular swelling had been noticed for eleven weeks. The patient made a good recovery from the operation. *See Surgical Reports,* vol. cliii, Case 36.

3459 Salivary Calculus.

A portion of a submaxillary salivary gland with its duct dilated, in which a calculus is lodged. A blue rod has been passed into the distal portion of the duct. Smaller calculi from the same case are seen at the bottom of the preparation glass.

John J., æt. 23, was admitted under Mr. Davies-Colley in 1890 for a swelling beneath the jaw on the right side, and a hard mass which could be felt on the floor of the mouth at the situation of the opening of Wharton's duct. Six months previously a calculus had been removed from the same duct. The submaxillary gland was excised at the second operation. *See Surgical Reports,* vol. cxlvii, Case 111.

3471 Fibrous Epulis.

The stump of a lateral incisor tooth from the upper jaw, surrounded by a firm lobulated growth about the size and shape of the terminal phalanx of an index finger. Histologically the tumour consists of fibrous tissue.

Removed from a man, æt. 31, who had noticed the growth for about one year. The teeth of the upper jaw were decayed, and the gum was thickened from chronic inflammation produced by the irritation of a plate for artificial teeth. *See Note-Book,* No. 3, p. 11.

Presented by Mr. GOLDING-BIRD, 1889.

3487 Tuberculous Infiltration of the Tongue.

A tongue, with the larynx and upper part of the trachea, showing at its root considerable swelling beneath the mucous membrane. The greater part of the epiglottis and the mucous membrane lining the larynx and trachea are destroyed by ulceration. Histological examination of the base of the tongue shows that the mucous membrane is intact, but that the tissue beneath it is infiltrated with a deposit of tuberculous nodules crowded with the specific bacilli.

Alfred M., æt. 42, was admitted under Dr. Taylor for phthisis with ulceration of the larynx. During the three weeks before his death a rapid destruction of the epiglottis was observed. At the autopsy there was recent and chronic phthisis of both lungs with numerous tuberculous ulcers in the intestine. *See Insp.*, 1889, No. 470.

3564 Traumatic Stenosis of the Œsophagus.

An œsophagus and stomach showing the lower three inches of the gullet uniformly narrowed so as barely to admit a No. 5 catheter. The walls of the constricted portion are thickened by inflammatory material, and the œsophagus above is somewhat dilated. On the reverse of the specimen may be seen the opening left after gastrotomy, around which there are several lacerations of the serous coat of the stomach produced by the giving way of the stitches which attached the viscus to the abdominal wall.

Percy H., æt. 11, was admitted under Mr. Symonds for difficulty in swallowing, having three months previously drunk a mouthful of carbolic acid. Before and after his admission to the hospital attempts were made to relieve the obstruction by the passage of bougies and by intubation. Eventually gastrotomy was performed, and ten days later the patient died from peritonitis. *See Insp.*, 1890, No. 75.

3567 Foreign Body in the Œsophagus.

The lower end of an œsophagus in which a vulcanite tooth-plate is impacted.

Edward W., æt. 44, was admitted under Mr. Jacobson seven weeks after he had swallowed a tooth-plate. Gastrotomy was performed, but it was found to be impossible to dislodge the foreign body. *See Insp.*, 1889, No. 199.

3570 Acute Inflammation of the Œsophagus.

An œsophagus denuded of epithelium throughout its length. In the upper third the condition is due to post-mortem solution, the limit of which is clearly defined at the junction of the pharynx and œsophagus. Below this part the walls of the tube are somewhat thickened, and its inner surface is rough and shaggy. Histological examination of the lowest portion of the œsophagus shows the submucous tissue to be much thickened and densely infiltrated with small round cells. The blood-vessels are dilated and filled with red corpuscles; the epithelium has disappeared.

John McL., æt. 78, was admitted under Mr. Davies-Colley for a fracture of the femur and other injuries received by being run over. For three days after admission he frequently vomited "coffee-ground" material, which gave the reaction for blood. He died ten days after the accident. At the autopsy blood-stained mucus was found in the œsophagus, but no food. The stomach was healthy. *See Insp.*, 1890, No. 36.

3587 Epitheliomatous Tumour of the Œsophagus.

A portion of the œsophagus showing the lumen of the tube to be obstructed by a conical tumour about two and a half inches long and one inch and a half in diameter. The upper part of the tumour is free and presents a ragged sloughy surface, while the lower half has a broad attachment to the posterior wall of the œsophagus. Immediately below the tumour, at the level of the bifurcation of the trachea, the wall of the gullet is much thickened by infiltration by growth and its mucous membrane is ulcerated. On the reverse of the specimen the growth is seen to have invaded and destroyed the muscular coat. There is a slight dilatation of the œsophagus above the obstruction. Histologically the tumour is a squamous-celled epithelioma.

John T., æt. 55, was admitted under Mr. Symonds for dysphagia of five months' duration. For nine weeks he had been able to swallow only liquid food. Unsuccessful attempts were made to pass bougies down the œsophagus. At the autopsy broncho-pneumonia was found. There were no secondary deposits. *See Insp.*, 1890, No. 369; and *Trans. Path. Soc.*, vol. xlii, p. 89.

3604 Œsophagus perforated by an Aneurysm.

An œsophagus with the lower end of the trachea and the descending portion of the arch of the aorta. Just below the level of the bifurcation of the trachea there is a small clean-cut opening in the wall of the œsophagus communicating with an aneurysm of the aorta. The sac of the aneurysm is lined with laminated blood-clot, which partly occludes the perforation. On the opposite wall of the œsophagus at the corresponding level there is a deep oval ulcer.

From a man who was carried to the hospital dead, having immediately before brought up a large quantity of blood. At the autopsy the stomach was found to contain more than a pint of blood-clot. *See Insp.*, 1890, No. 393.

LIST OF SPECIMENS ADDED TO THE MUSEUM OF COMPARATIVE ANATOMY.

By H. J. CAMPBELL.

Longitudinal Section of Dog's Skull showing Brain in situ.

Wet Preparation of Skull and Visceral Arches of Dog-fish.

Wet Preparation of Skeleton of a Frog (Rana Guppyi) from the Solomon Islands.

Liver of Fournier's Capromys (Capromys pilorides).

LIST OF SPECIMENS ADDED TO THE MUSEUM OF COMPARATIVE PATHOLOGY.

BY H. J. CAMPBELL.

These specimens were obtained from animals dying in the Zoological Society's Gardens, by the kind permission of the Society and of Mr. Beddard.

1782 Intestinal Obstruction due to Ascarides in a Crane (Grus communis).

Windows have been cut in the small intestine to show the ascarides *in situ;* the intestine above was not dilated, and liquid fæces were found in the intestine below the partial obstruction.

1783 Tapeworms (Solenophorus megalocephalus) in Intestine of a Python.

The intestine was crowded with about fifty worms attached to the mucous membrane over a surface about eight inches in extent.

1784 Tubercular Nodules in Large Intestine and Cæcum of a Rhea Americana.

The preparation also shows an intussusception of the cæcum, one of the nodules having been caught by the contraction of the cæcum below, and having dragged downward in its turn the part of the cæcum to which it was attached.

1785 Intussusception of Large Intestine in a Boa Constrictor.

The lower end of the intussuscepted gut was three inches from the anus. The intestine above the intussusception was much dilated.

1787 Encapsuled Calculus in Apex of Kidney of Horse.

The calculus consists of pure carbonate of lime; the cyst has been opened so as to show the calculus within; all trace of kidney tissue has disappeared from the capsule covering the calculus.

1790 Tubercular Caseous Nodules in Liver and Spleen of a Crane (Grus communis).

1791 Tubercular Disease of the Spleen in a Rhesus Monkey (Macacus rhesus).

The animal died from general tuberculosis; the spleen shows several caseous abscesses.

Foreign Body in Œsophagus and Stomach of a White-tailed Eagle (Haliætus albicilla).

1792 Tubercular Disease of Lungs and Caseous Glands in a Rhesus Monkey (Macacus rhesus).

The primary disease appears to have been in the glands, the lungs having been secondarily affected by direct infection.

1793 Small-celled Growth in Lung of an Upland Goose (Bernicla magellanica).

The character of the growth was doubtful; it looks enchondromatous, and is very hard, but microscopically it shows no trace of cartilage.

LIST

OF

GENTLEMEN EDUCATED AT GUY'S HOSPITAL

WHO HAVE PASSED THE

EXAMINATIONS OF THE SEVERAL UNIVERSITIES, COLLEGES, &C. &C.,

IN THE YEAR 1890.

University of Oxford.

Examination for the Degree of Doctor of Medicine.

G. H. Pennell.

University of Cambridge.

Third Examination for the Medical and Surgical Degrees.

Part II.

E. S. Blaker.
B. B. Connolly.
A. E. Durham.
D. S. Long.
E. J. D. Mitchell.
E. M. Pilcher.
G. Pinder.
J. W. Russell.
D. W. Samways.
H. A. Smith.
C. R. A. Sutton.
F. G. Swayne.
F. Wellford.
W. Winslow.

Part I.

W. Carling.
M. Craig.
W. H. Fisher.
A. H. Godson.
D. S. Long.
C. G. Roberts.
D. W. Samways.
H. A. Smith.
C. R. A. Sutton.
F. G. Swayne.
F. M. Turner.
F. Wellford.
W. Winslow.

Second Examination for the Medical and Surgical Degrees.

Part I.

Pharmaceutical Chemistry.

H. B. Bolus.
C. E. Cooper.
C. Trouncer.

Part II.

Anatomy and Physiology.

W. E. Alston.
C. E. Cooper.
F. N. Day.
G. H. Field.
W. R. Grove.
W. J. Harris.
G. Lawrence.
E. Ransome.
A. L. Roper.
A. Shillitoe.
G. F. Still.

First Examination for the Medical and Surgical Degrees.

Part I.

Chemistry and Physics.

C. D. Fothergill.
F. A. Godson.
A. L. Roper.

Part II.

Biology.

F. A. Godson.

University of London.

Examination for the Degree of Doctor of Medicine.

E. H. Starling.

Obtained the number of Marks qualifying for the Gold Medal.

H. J. Campbell.
G. A. Carpenter.
E. Moss.
A. Parkin.
J. Lloyd Roberts.
T. Wilson Smith.

Examination for the Degree of Master of Surgery.

R. D. Mothersole.
A. H. Tubby.

Examination for the Degree of Bachelor of Surgery.

First Division.

F. W. Hall.

Obtained Honours.

A. E. Norburn.
J. S. Richards.

Second Division.

J. H. Bryant.
J. Fawcett.
T. F. Ricketts.
T. G. Stevens.
H. W. Webber.

Final Examination for the Degree of Bachelor of Medicine.

May.

First Division.

J. McD. Gill.

Second Division.

H. J. Blackler. | S. B. De Mesquita.

October.

First Division.

J. H. Bryant

Obtained the Gold Medal and First-Class Honours in Medicine, also First-Class Honours in Forensic Medicine.

P. C. Evans

Obtained First-Class Honours in Medicine and in Obstetric Medicine, and Honours in Forensic Medicine.

J. S. Richards

Obtained Honours in Medicine and Obstetric Medicine.

F. W. Hall, | J. Robertson,

J. Fawcett,

Obtained Honours in Obstetric Medicine.

T. G. Stevens

Obtained Honours in Forensic Medicine.

A. E. Norburn. | T. F. Ricketts.

H. W. Webber.

Second Division.

R. A. Bindley.

Intermediate Examination in Medicine.

January.

First Division.

C. E. Salter.

Second Division.

W. Bligh. | S. G. Floyd.

Excluding Physiology.

A. E. Tebb.

July.

Honours Examination.

C. S. Pantin

Obtained Honours in Anatomy, Physiology, and Histology.

F. G. Hopkins

Obtained the Exhibition and Gold Medal in Organic Chemistry and Honours in Materia Medica and Pharmaceutical Chemistry.

A. J. Sharp

Obtained Honours in Physiology and Histology, and also in Materia Medica and Pharmaceutical Chemistry.

Pass List.

Second Division.

F. J. Coleman. | E. S. Dukes.

W. J. Johnson.

Excluding Physiology.

A. H. Card.
F. Hazell.
W. H. Jewell.

Physiology only.

A. E. Tebb.

Examination for the Degree of Bachelor of Science.

First Division.

F. G. Hopkins.

Intermediate Examination in Science.

First Division.

E. J. Parry.

Preliminary Scientific (M.B.) Examination.

January.

Entire Examination.

First Division.

S. P. James.

Second Division.

F. J. H. Cann.
A. Miller.

Two Subjects of the Examination (*Chemistry and Physics*).

A. C. Hovenden.
G. S. Hovenden.

One Subject of the Examination (*Biology*).

J. Robertson.

July.

Honours Examination.

J. Hora

Obtained Honours in Inorganic Chemistry.

A. Salter

Obtained Honours in Botany.

Pass List.

Entire Examination.

First Division.

W. S. Handley.
E. T. Scowby.
F. J. Steward.

Second Division.

E. Coleman.
E. S. Hall.
J. H. Horton.
E. Moore.
P. N. Vellacott.

Two Subjects of the Examination (*Chemistry and Experimental Physics*).

J. Moore.
H. J. Spon.
J. R. Steinhaeuser.

(Chemistry and Biology.)

C. H. Hunter.

One Subject of the Examination (*Biology*).

O. Bevan.
W. E. Cross.
C. J. Harnett.
D. Rice
W. W. Waymark.
H. M. Wise.

(Chemistry.)

W. G. Mumford.

University of Durham.

Examination for the Degree of Doctor of Medicine.

E. W. Simmons.
J. Utting.

Final Examination for the Degree of Bachelor of Medicine.

W. J. Scott.

Final Examination for the Degree of Bachelor of Surgery.

W. J. Scott.

Second Examination for the Degree of Bachelor of Medicine.

J. W. H. Eyre.

Examination for the Degree in Sanitary Science.

J. A. Marsden.

Naval Medical Service.

London.

R. H. J. Browne.
P. N. Randall.

Indian Medical Service.

London.

B. das Basu.

Royal College of Physicians.

Fellowship.

Henry Ashby.
E. Clifford Beale.

Final Examination for the Licence.

January.

F. W. S. Davies.
J. A. Fox.
J. McD. Gill.
E. B. Harris.
H. V. Hickman.
D. T. Jones.
R. G. P. Lansdown.
H. B. Perkins.
F. R. H. Potts.
J. H. Prall.
W. J. Scott.
C. Spurrell.

April.

T. F. Christal.
A. C. Elliman.
J. Fawcett.
F. F. F. Foster.
F. W. Hall.
V. J. Hodgson.
C. Legg.
F. P. Sarjant.
W. S. M. Smith.
H. B. Statter.
H. W. Whyte.
W. W. Wingate.

July.

J. H. Bryant.
N. B. Clowes.
H. Hodgson.
B. W. Hogarth.
C. McGregor.
E. J. D. Mitchell.
R. G. Pollock.
C. J. Prime.
J. S. Richards.
T. G. Stevens.
J. M. Thorne.
F. G. Vicars.
H. W. Webber.
A. S. Wohlmann.
F. S. Wood.

October.

F. W. Farr.
H. W. John.
G. H. Knapp.
N. L. Richards.
H. A. Smith.

Royal College of Surgeons.

Final Examination for the Fellowship.

W. L. Blight.
A. T. Brown.
H. E. Cuff.
R. D. Mothersole.
A. Parkin.
G. B. Smith.

First Examination for the Fellowship.

D. M. Beddoe.
A. Chaning-Pearce.
W. J. Harris.
H. M. Jordan.
G. Lawrence.
J. B. Leathes.
R. H. Luce.
C. S. Pantin.
D. W. Samways.
A. J. Sharp.

Final Examination for the Membership.

January.

F. W. S. Davies.
J. A. Fox.
J. McD. Gill.
E. B. Harris.
H. V. Hickman.
D. T. Jones.
R. G. P. Lansdown.
H. B. Perkins.
J. H. Prall.
W. J. Scott.
C. Spurrell.

April.

T. F. Christal.
A. C. Elliman.
J. Fawcett.
F. F. F. Foster.
F. W. Hall.
V. J. Hodgson.
C. Legg.
F. P. Sarjant.
W. S. M. Smith.
H. B. Statter.
H. W. Whyte.
W. W. Wingate.

July.

J. H. Bryant.
N. B. Clowes.
H. Hodgson.
B. W. Hogarth.
C. McGregor.
E. J. D. Mitchell.
R. G. Pollock.
C. J. Prime.
J. S. Richards.
T. G. Stevens.
J. M. Thorne.
F. G. Vicars.
H. W. Webber.
A. S. Wohlmann.
F. S. Wood.

October.

H. W. John.
G. H. Knapp.
N. L. Richards.
H. A. Smith.

MEDALLISTS AND PRIZEMEN, 1889-90.

The Treasurer's Gold Medal for Clinical Medicine.

Arthur Ellis Durham, London.
John Henry Bryant, Ilminster. *Proxime accessit.*

The Treasurer's Gold Medal for Clinical Surgery.

Frederick William Hall, Sydney.

Gurney Hoare Prize for Clinical Study.

Robert Devereux Mothersole, Colchester.

Beaney Prize for Pathology.

John Fawcett, Westgate-on-Sea.

Golding-Bird Prize and Medal.

Thomas George Stevens, Newington Green.

Michael Harris Prize for Anatomy.

Robert Henry Ritchie, New South Wales.

The Arthur Durham Prizes for Dissection.

First Year's Students.

Henry William Collier, Peckham, S.E.

Second Year's Students.

John Beresford Leathes, Rochester.

Fourth Year's Students.

Bertram Whewell Hogarth, Morecambe, First Prize, £25.
John Henry Bryant, Ilminster, Second Prize, £10.
Harold Hodgson, St. John's Wood, Certificate.

Third Year's Students.

Alfred Theodore Rake, Fordingbridge, First Prize, £25.
Thomas Holmes, Lancaster, Second Prize, £10.
Thomas Benjamin Philip Davies, Milford Haven, Certificate.

Second Year's Students.

Arthur John Sharp, Bromley, Kent, First Prize, £25.
Charles Satchell Pautin, Blackheath, Second Prize, £10.
Frederick Gowland Hopkins, Enfield, Certificate.
William John Johnson, Shefford, Certificate.
Francis Jordan Coleman, Hunmanby, Yorks, Certificate.

First Year's Students.

Arthur Henry Leete, Wellingborough, First Prize, £50.
John Alexander Howard, Canonbury, Second Prize, £25.
Guy Eugene Manning, Hammersmirh, Certificate.
Leopold Ernest Valentine Every Clayton, Nelson, Lancashire, Certificate.

Open Scholarships in Arts.

Henry Percival Ferraby, Bedford, 100 Guineas.
George Frederick Still, Tollington Park, 50 Guineas.

Open Scholarships in Science.

Francis James Steward, Old Charlton, 125 Guineas.
Alfred Salter, Blackheath, 50 Guineas.

Physical Society.

Session 1890–91.

Honorary President.—SAMUEL WILKS, M.D., LL.D., F.R.S.

Presidents.

Messrs. W. F. Colclough, B.A., A. E. Durham, B.A., H. E. Durham, B.A., J. Fawcett, J. M. Gill, M.B., F. W. Hall, H. V. Hickman, F. G. Hopkins, A. E. Norburn, A. T. Rake, A. J. Sharp, G. B. Smith, M.B., F. G. Swayne, M.B., F. G. Vicars, C. F. Wakefield, H. W. Webber.

Exhibits Committee.

Messrs. W. F. Colclough, B.A., J. M. Gill, F. G. Hopkins, C. F. Wakefield.

Honorary Secretaries.

L. A. DUNN, M.S., LAURISTON E. SHAW, M.D.

PRIZEMEN FOR THE SESSION 1889–90.

The Treasurer's Prize of £8 for Essay on "Heredity" was awarded to Mr. A. J. Sharp.

The Society's Prizes: First, £7, was awarded to Mr. F. G. Hopkins, B.S., for his Paper on "The Chemical Genesis of Physiological Pigments;" Second, Mr. H. E. Durham, £3, for his Paper on "Some Points which concern Predisposition and Immunity in Disease." An extra Prize of £2 2*s*. was given to Mr. F. Colclough for his Essay on "Heredity."

CLINICAL APPOINTMENTS HELD IN THE YEAR 1890.

HOUSE PHYSICIANS.

H. E. Cuff.
A. D. Fripp.
A. E. Norburn.
J. W. Russell.
H. V. Hickman.
A. T. Brown.
A. E. Durham.
J. McD. Gill.

HOUSE SURGEONS.

A. T. Brown.
A. T. MacConkey.
S. W. MacIlwaine.
J. W. Smith.
H. J. Holman.
P. Paget.
F. J. Nisbett.

OBSTETRIC RESIDENTS.

A. E. Palmer.
J. W. Roberts.
C. M. Kitching.
H. J. Holman.
H. A. Smith.
A. L. D. Meares.
H. V. Hickman.
R. G. P. Lansdown.
G. H. S. Daniell.
J. McD. Gill.
J. W. Russell.
C. Spurrell.
W. J. Scott.

SURGEONS' DRESSERS.

R. G. Pollock.
C. R. A. Sutton.
R. S. Freeland.
J. H. Bryant.
F. H. Brown.
H. P. O. Manning.
J. Young.
W. S. M. Smith.
E. H. Baldock.
W. G. Rogers.
E. G. March.
J. M. Thorne.
B. W. Hogarth.
H. W. Webber.
A. E. Kelsey.
F. A. Osborn.
F. Colclough.
E. H. Cartwright.
G. H. Knapp (one month).
H. M. Jordan.
C. G. Roberts.
H. W. Whyte.
V. J. Hodgson.
A. Thomas.
W. Winslow.
W. Carling.
F. Wellford.
J. W. F. Jewell.
D. W. Samways.
W. K. Steele.
A. C. Elliman.
A. S. Wohlmann.
T. G. Stevens.
E. M. Dobinson.
H. W. John.
F. D. Lumley.
F. E. Williams.
W. A. Bryant (two months).
W. E. S. Cobb.
C. D. H. Ralph.
H. B. Rygate.
H. Hodgson.
D. T. Jones.
E. G. March.
F. G. Vicars.
W. Carling.
A. Thomas.
W. H. Dixon.
G. Pender-Smith.
H. A. Bryant.
D. S. Long.
A. H. Meadows.
E. J. Lang.
W. J. Robertson.
A. E. Tebb.
E. Cornish.
H. St. J. Fraser (one month).

CLINICAL ASSISTANTS.

J. Fawcett.
J. W. Russell.
A. E. Durham.
H. A. Smith.
A. S. Wohlmann.
T. G. Stevens.
E. S. Blaker.
E. W. Wheatcroft.
J. W. Roberts.
J. H. Bryant.
A. H. Meadows.
F. H. Brown.
B. W. Hogarth.
E. J. D. Mitchell.
S. J. Roberts.
A. T. White.
H. W. Webber.
W. Wingate.
J. Young.
W. S. M. Smith.
F. G. Swayne.
D. S. Long.
C. R. A. Sutton.
E. M. Pilcher.

DRESSERS IN THE EYE WARDS.

F. S. Wood.
R. G. P. Lansdown.
J. McD. Gill.
F. W. Hall.
J. Fawcett.
H. A. Smith.
C. D. H. Ralph.
N. B. Clowes.
F. G. Swayne.
J. P. Pendlebury.
G. H. S. Daniell.
F. D. Lumley.
G. H. Knapp.
W. D. Spurrell.
A. E. Kelsey.
J. M. Thorne.
N. L. Richards.
C. Spurrell.
J. S. Richards.
C. R. A. Sutton.
R. S. Freeland.
C. G. Roberts.
G. W. Featherstone.

DENTAL SURGEONS' DRESSERS.

F. J. Nisbett.
F. W. Ta Bois.
J. M. Thorne.
E. P. Donston.
C. Spurrell.
H. J. L. Wales.
V. H. Barr.
J. Young.
W. E. S. Cobb.
J. S. Richards.
H. Richardson.
W. D. Spurrell.
F. D. Lumley.
A. G. Buchanan.
W. E. Alston.
H. G. Howitt.
F. A. Robinson.
P. Harrison.
H. Stoner.
T. S. Robson.
W. H. Dixon.
R. G. James.
W. K. Steele.
R. Apthorpe.

MEDICAL WARD CLERKS.

E. Cornish.
E. H. Baldock.
M. Jenkins.
W. A. Higgs.
H. G. Biddle.
F. D. Lumley.
E. M. Rooke.
D. W. Samways.
A. H. Meadows.
D. S. Long.
H. A. Bryant.
W. K. Steele.
F. E. Williams.
E. H. Cartwright.
R. L. Wason.
S. J. Roberts.
E. E. Bensley.
H. E. Worthington.
T. I. Mills.
C. Braithwaite.
E. E. B. Landon.
W. A. Haslam.
H. B. Wilkinson.
P. M. May.
W. D. Loveday.
H. St. J. Fraser.
A. T. Rake.
T. Holmes.
T. B. P. Davies.
A. Reeves.
A. T. Jago.
R. Gillbard.
C. E. Salter.
G. Sichel.
W. T. B. Donnelly.
T. R. Taylor.
C. M. Fleury.
C. E. Moffatt.
A. W. Sheen.
A. M. Daldy.
E. T. E. Hamilton.
G. Pinder.
C. E. Morice.
H. E. Durham.
J. E. Coulson.

ASSISTANT SURGEONS' DRESSERS.

E. E. Bensley.
A. L. Allworth.
W. Poole.
S. J. Roberts.
J. E. Coulson.
G. Pinder.
H. K. Rayson.
J. H. Barnes.
H. Hodgson.
E. H. Cartwright.
G. F. Birdwood.
C. R. B. Spencer.
H. B. Bolus.
J. J. Browne.
A. M. Daniel.
W. H. B. Hall.
O. C. Worthington.
C. B. Braithwaite.
P. M. May.
H. A. Bryant.
A. H. Godson.
C. N. Thomas.
W. H. Fisher.
P. Lord.
F. C. Young.
A. Reeves.
A. T. Jago.
H. G. Biddle.
R. Gillbard.
W. S. Frith.
C. G. Morice.
J. R. R. Pollock.
F. W. Wilson.
E. G. Evans.
F. P. S. Cresswell.
H. E. Worthington.
H. B. Wilkinson.
W. D. Loveday.
G. Sichel.
C. E. Salter.
A. W. Sheen.
F. M. Turner.
J. H. Muncaster.
C. E. Pollock.
V. H. Barr.
J. R. Thomas.
W. Colclough.
C. C. Elliott.
W. H. Dixon.
T. H. Evans.
E. E. B. Landon.
S. Croneen.
W. A. Haslam.
H. St. J. Fraser.
T. Holmes.

DRESSERS IN THE SURGERY.

H. E. Worthington.
C. B. Braithwaite.
C. E. Salter.
N. Instone.
E. E. B. Landon.
A. M. Daldy.
E. G. Evans.
C. C. Elliott.
W. D. Loveday.
C. M. Fleury.
H. St. J. Fraser.
P. M. May.
F. M. Turner.
A. M. Daniel.
J. R. Evans.
F. C. Stearn.
C. E. Moffatt.
A. T. Rake.

AURAL SURGEONS' DRESSERS.

J. Young.
R. G. James.
W. K. Steele.
G. W. Featherstone.
C. R. Graham.
W. G. Beyts.
W. Winslow.
C. S. Spong.
H. Richardson.
G. Muckeson.
A. Shillitoe.
H. V. Hickman.
J. W. Russell.
W. D. Spurrell.

ASSISTANT PHYSICIANS' CLERKS.

B. W. Hogarth.
W. Winslow.
T. I. Mills.
C. B. Braithwaite.
F. Colclough.
D. W. Samways.
H. E. Worthington.
F. W. Hall.
C. E. Salter.
H. L. E. Wilks.
A. R. Lacey.
H. B. Rygate.
W. B. Donnelley.
A. M. Daldy.
F. G. Saffrey.
D. S. Long.
G. H. Knapp.
H. B. Wilkinson.
E. T. E. Hamilton.
A. T. White.
C. E. Pollock.
T. H. Evans.
J. O. Williams.
W. H. B. Hall.
F. D. Lumley.
F. E. Williams.
E. H. Cartwright.
V. H. Barr.

POST-MORTEM CLERKS.

B. W. Hogarth.
A. C. Elliman.
F. Colclough.
H. E. Durham.
E. M. Pilcher.
J. H. Muncaster.
F. G. Saffrey.
A. A. Grosvenor.
E. T. E. Hamilton.
H. L. E. Wilks.
J. S. Barnes.
A. T. White.
E. W. Wheatcroft.
A. L. Roper.
J. D. Thomas.
H. B. Bolus.
T. I. Mills.
J. R. R. Pollock.
W. Poole.

OBSTETRIC WARD CLERKS.

F. G. Saffrey.
W. Wingate.
W. Carling.
A. E. Norris.
W. E. Sturges-Jones.
G. Mackeson.
G. W. A. Mitchell.
H. Meyrick-Jones.
J. C. Bates.
F. A. Osborn.
N. L. Richards (six weeks).
A. Allport.

OBSTETRIC OUT-PATIENTS' CLERKS.

W. Winslow.
F. G. Saffrey.
T. H. Evans.
F. M. Turner.
F. W. Wellford.
A. Thomas.
F. A. Osborn.
F. Colclough.
G. T. Birdwood.
H. J. L. Wales.
F. C. Stearn.
S. J. Roberts.
J. Young.
R. H. Luce.
W. E. Alston.
A. G. Buchanan.
J. H. Busteed.
E. E. W. Roe.

DRESSERS IN THE THROAT DEPARTMENT.

P. Purnell.
V. J. Hodgson.
E. M. Dobinson.
A. B. Hudson.
F. G. Saffrey.
R. E. Williams.
F. W. Hall.
F. G. Vicars.
A. E. Durham.
J. W. Russell.
J. Fawcett.
F. S. Wood.
J. S. Richards.
J. H. Bryant.
G. H. Knapp.
H. E. Richardson.

EXTERN OBSTETRIC ATTENDANTS.

A. Thomas.
E. G. March.
F. M. Russell.
A. Allport.
F. E. Williams.
A. L. Allworth.
V. H. Barr.
C. E. Pollock.
H. A. Bryant.
W. S. Frith.
F. C. Young.
T. S. Jackson.
A. M. Daniel.
T. I. Mills.
E. H. Cartwright.
F. Colclough.
J. R. R. Pollock.
H. J. Wales.
H. G. Biddle.
A. H. Meadows.
T. H. Evans.
E. G. Evans.
H. P. Barker.
D. W. Samways.
F. M. Turner.
F. C. Stearn.
E. G. Lang.
A. T. White.
G. A. Skinner.
H. C. Sharp.
J. D. Thomas.
E. E. Bensley.
J. W. Eastment.
E. W. Wheatcroft.
E. Cornish.
W. A. Higgs.
E. H. Baldock.
H. Hodgson.
W. G. Rogers.
J. W. F. Jewell.
H. L. E. Wilks.
E. Huntley.
A. H. Godson.
J. O. Williams.
A. G. Buchanan.
A. E. Norris.
F. W. Wilson.
S. G. Floyd.
D. A. W. Martin.
F. P. S. Cresswell.

SURGICAL WARD CLERKS.

P. Lord.
T. I. Mills.
W. A. Haslam.
R. Gillbard.
G. Sichel.
H. W. Wilkinson.
F. W. Wilson.
A. H. Godson.
W. H. Fisher.
C. N. Thomas.
W. S. Frith.
F. C. Young.
A. W. Sheen.
E. T. E. Hamilton.
F. P. S. Cresswell.
R. L. Wason.
W. T. B. Donnelley.
T. Holmes.
T. R. Taylor.
C. G. Morice.
J. W. Taylor.
R. E. G. Phillips.
M. Craig.
C. E. Cooper.
E. S. Dukes.
T. S. Jackson.
A. Allport.
H. J. L. Wales.
E. E. Frazer.
C. M. Fleury.
F. C. Stearn.
T. S. Robson.
J. S. Barnes.
J. R. Evans.
A. M. Daniel.
G. T. Birdwood.
T. H. Evans.
E. P. H. Lulham.
J. O. Williams.
A. G. Buchanan.
J. D. Thomas.
R. H. Luce.
F. P. Bush.
F. A. Robinson.
H. A. Hinds.
C. S. Pantin.
H. Cooper.
J. H. Joyce.
J. H. Godson.
W. J. Johnson.
E. Huntley.
S. G. Floyd.
T. D. Lister.
W. Bligh.
S. Croneen.
A. M. Jones.
J. J. E. Biggs.
J. H. Compton.
E. E. W. Roe.
J. W. H. Eyre.
W. Poole.
A. L. Roper.
H. C. Sharp.
S. Cresswell.
C. N. Greenway.
G. H. Field.
W. E. Alston.
P. E. Bearblock.
E. Reid.
H. D. Eccles.
A. J. Sharp.
W. J. Harris.
F. M. Day.
F. J. Coleman.

ASSISTANT SURGEONS' CLERKS.

R. G. James.
J. W. F. Eyre.
H. C. Sharp.
F. C. Stearn.
J. R. Evans.
H. J. L. Wales.
F. Hazell.
E. S. Dukes.
C. M. Fleury.
A. T. Rake.
C. E. Carpmael.
E. L. Adams.
A. F. Clarke.
T. S. Robson.
W. H. Jewell.
E. Ransome.
T. H. Evans.
A. M. Daldy.
E. L. Adams.
G. Pinder.
G. T. Birdwood.
W. C. C. Park.
R. H. Luce.

DENTAL SCHOOL.

APPOINTMENTS HELD DURING THE YEAR 1890.

ASSISTANT DENTAL HOUSE SURGEON.

F. W. Ta Bois.

CURATORS OF LABORATORY.

H. O. W. Harris.
P. Harrison.

DRESSERS IN THE EXTRACTION ROOM.

E. P. Donston.
S. R. Apthorpe.
C. Read.
H. B. Stoner.
F. W. Ta Bois.
P. Harrison.
H. G. Howitt.

DRESSERS IN THE GAS ROOM.

H. O. W. Harris.
M. F. Hopson.
P. Harrison.
H. B. Stoner.
F. Haynes.
F. W. Ta Bois.
J. S. Sewill.

LIST

OF

GENTLEMEN EDUCATED AT GUY'S HOSPITAL

WHO HAVE PASSED THE

EXAMINATIONS OF THE SEVERAL UNIVERSITIES, COLLEGES,

&c. &c.,

IN THE YEAR 1891.

University of Oxford.

Final Examination for the Degrees of Bachelor of Medicine and Surgery.

F. H. Burton-Brown.

University of Cambridge.

Examination for the Degree of Doctor of Medicine.

B. B. Connolly.

Third Examination for the Degree of Bachelor of Medicine.

Part II.

H. B. Bolus.
W. Carling.
F. Colclough.
M. Craig.
H. E. Durham.
T. H. Evans.
W. H. Fisher.
F. J. Gillibrand.
A. H. Godson.
F. M. Turner.

Part I.

A. M. Daniel.
H. E. Durham.
T. H. Evans.
J. H. Joyce.
G. Pinder.
C. N. Thomas.
J. D. Thomas.
F. C. Young.

Second Examination for the Degree of Bachelor of Medicine.

Part II.

C. S. Bond.
L. C. Burrell.
E. R. Delbruck.
W. L. Garner.
T. P. King.
E. A. Peters.
H. M. Stewart.

Part I.

C. L. Hopkins.

B. W. M. A. Key.

E. A. Peters.

University of London.

Examination for the Degree of Doctor of Medicine.

E. H. Brock.
J. H. Bryant.
H. E. Cuff.
P. C. Evans.
J. Fawcett.
J. McD. Gill.
F. W. Hall.
C. McG. Kitching.
W. J. Maillard.
R. D. Mothersole.
J. S. Richards.
T. F. Ricketts.
J. Robertson.
A. Scott.
H. W. Webber.

Examination for the Degree of Bachelor of Surgery.

First Division.

W. G. Rogers

Obtained First-Class Honours.

T. B. P. Davies,

H. Hodgson,

Obtained Second-Class Honours.

A. T. Rake,

A. S. Wohlmann,

Obtained Third-Class Honours.

B. W. Hogarth.

Second Division.

J. Lloyd Roberts

Obtained First-Class Honours.

S. Bueno de Mesquita.
C. E. Salter.
A. E. Tebb.
A. Thomas.
J. Young.

Final Examination for the Degree of Bachelor of Medicine,

May.

First Division.

W. G. Rogers.

Second Division.

H. V. Hickman.

October.

First Division.

T. B. P. Davies

Obtained Second-Class Honours in Medicine, and Third-Class Honours in Forensic Medicine.

B. W. Hogarth

Obtained Third-Class Honours in Medicine, and Second-Class Honours in Obstetric Medicine and in Forensic Medicine.

A. T. Rake

Obtained Second-Class Honours in Medicine, in Obstetric Medicine, and in Forensic Medicine.

H. Hodgson. | A. S. Wohlmann.
J. Young.

Second Division.

A. Thomas

Obtained Third-Class Honours in Obstetric Medicine.

C. E. Salter. | F. P. Sarjant.
A. E. Tebb.

Intermediate Examination in Medicine.

January.

Second Division.

R. C. Kirkby. | T. D. Manning.

Excluding Physiology.

Second Division.

E. L. Adams. | A. P. Allan.

Physiology only.

Second Division.

A. H. Card. | W. H. Jewell.
W. C. C. Park.

July.

Honours Examination.

J. A. Howard

Obtained the Exhibition and Gold Medal in Organic Chemistry, and Honours in Materia Medica and Pharmaceutical Chemistry.

A. H. Leete

Obtained Honours in Physiology and Histology.

L. E. V. Every-Clayton

Obtained Honours in Materia Medica and Pharmaceutical Chemistry.

Pass List.

First Division.

G. E. Manning.

Second Division.

E. H. Drake. | G. L. Eastes. | P. McK. C. Wilmot.

Excluding Physiology.

Second Division.

W. E. de Korte.

Physiology only.

Second Division.

E. L. Adams.
A. P. Allan.
F. Hazell.

Intermediate Examination in Science.

Pass List.

First Division.

G. D. Lander.

Preliminary Scientific (M.B.) Examination.

January.

Entire Examination.

Pass List.

First Division.

T. H. Green.

Chemistry and Physics only.

W. E. Cross.
A. Earnshaw.
C. J. Harnett.
D. Rice.
H. M. Wise.

Biology only.

C. H. Murray.
D. L. Smith.
H. J. Spon.
J. R. Steinhäuser.
T. M. Thomas.
S. Whicher.

Chemistry only.

H. F. Turner.

July.

Entire Examination.

Second Division.

H. P. Ferraby.
P. H. Haylett.
J. Howell.
D. B. Todd.

Chemistry and Physics only.

C. H. Murray.
D. L. Smith.
S. Whicher.

Biology only.

J. Moore.

University of Durham.

Examination for the Degree of Doctor of Medicine.

L. W. Keiffenheim.
R. G. P. Lansdown.

Final Examination for the Degree of Bachelor of Medicine.

T. Carr (Honours).
J. V. Blachford.

Examination for the Degree of Bachelor of Surgery.

J. V. Blachford.

Second Examination for the Degree of Bachelor of Medicine.

A. G. Cooley.

First Examination for the Degree of Bachelor of Medicine.

Chemistry and Chemical Physics and Botany.

W. T. B. Donnelley.
E. R. Fothergill.
G. Jiminez.
W. F. Miller.
W. H. Peake.
F. W. Rowland.
W. J. Rowland.
F. Zumbado.

University of Aberdeen.

Final Examination for the Degree of Bachelor of Medicine and Surgery.

S. H. Burnett.

Naval Medical Service.

E. M. Dobinson.
A. E. Kelsey.
H. B. Marriott.
G. McGregor.
N. L. Richards.
E. E. P. Tindall.

Army Medical Service.

W. G. Beyts.
E. M. Pilcher.

Indian Medical Service.

A. T. Brown.
F. H. Burton-Brown.

Royal College of Physicians.

Examination for the Membership.

H. J. Campbell.
E. H. Starling.

Final Examination for the Licence.

January.

M. Adler.
W. C. Burt.
T. S. Coombe.
F. W. Foster.
R. S. Freeland.
H. B. Rygate.
D. W. Samways.
J. A. Ward.
W. Winslow.
J. Young.

April.

J. E. C. Allott.
W. G. Brett.
E. Cornish.
G. W. B. Featherstone.
A. T. Rake.
H. Richardson.
W. G. Rogers.
W. D. Spurrell.
A. Thomas.
H. L. E. Wilks.
J. C. Bates.

July.

H. S. Archdall.
M. Craig.
T. B. P. Davies.
E. M. Dobinson.
J. W. Emmet.
A. A. Grosvenor.
J. W. F. Jewell.
S. H. Lucy.
C. E. Salter.
J. S. Sewill.

October.

E. H. Baldock.
W. E. S. Cobb.
A. M. Daldy.
E. T. E. Hamilton.
A. B. Hudson.
E. J. Lang.
A. H. Meadows.
C. E. Pollock.
T. R. Taylor.

Royal College of Surgeons.

Final Examination for the Fellowship.

E. J. Morley.
J. Lloyd Roberts.

First Examination for the Fellowship.

D. M. Beddoe.
A. Chaning-Pearce.
W. J. Harris.
H. M. Jordan.
G. Lawrence.
J. B. Leathes.
R. H. Luce.
C. S. Pantin.
D. W. Samways.
A. J. Sharp.

Final Examination for the Membership.

January.

M. Adler.
W. C. Burt.
T. S. Coombe.
R. S. Freeland.
H. B. Rygate.
D. W. Samways.
W. Winslow.
J. Young.

April.

J. E. C. Allott.
W. G. Brett.
E. Cornish.
G. W. B. Featherstone.
A. T. Rake.
H. Richardson.
W. G. Rogers.
W. D. Spurrell.
A. Thomas.
H. L. E. Wilks.
J. C. Bates.

July.

H. S. Archdall.
M. Craig.
T. B. P. Davies.
E. M. Dobinson.
J. W. Emmet.
A. A. Grosvenor.
J. W. F. Jewell.
S. H. Lucy.
C. E. Salter.
J. S. Sewill.

October.

W. E. S. Cobb.
A. M. Daldy.
E. T. E. Hamilton.
A. B. Hudson.
E. J. Lang.
A. H. Meadows.
C. E. Pollock.
T. R. Taylor.

Final Examination for the Licence in Dental Surgery.

F. Haynes.
M. F. Hopson.
F. W. Ta Bois, jun.

MEDALLISTS AND PRIZEMEN, 1890-1.

The Treasurer's Gold Medal for Clinical Medicine.

John Henry Bryant, Ilminster.

The Treasurer's Gold Medal for Clinical Surgery.

John Henry Bryant, Ilminster.

Gurney Hoare Prize for Clinical Study.

Arthur Stanley Wohlmann, Hertford.

Beaney Prize for Pathology.

John Henry Bryant, Ilminster.

Golding-Bird Prize and Gold Medal.

Alfred Theodore Rake, Fordingbridge.

Michael Harris Prize for Anatomy.

Guy Eugene Manning, Hammersmith.

The Arthur Durham Prizes for Dissection.

First Year's Students.

James Henry Horton, Charlton, Prize £5.
Murray Parry Jones, Towyn, Certificate.
Charles Herbert Fagge, Lutterworth, Certificate.
Henry Alford Moffat, Grahamstown, Cape Colony, Certificate.

Second Year's Students.

Charles Satchell Pantin, Blackheath, Prize £15.
Edwin Shelton Roberts, Penygroes, Certificate.

Fourth Year's Students.

Thomas Benjamin Philip Davies, Milford Haven, } equal,
Alfred Theodore Rake, Fordingbridge, } £17 10*s*. each.
Arthur Mantell Daldy, Romford, Certificate.

Third Year's Students.

Arthur John Sharp, Bromley, Kent, First Prize, £25.
William John Johnson, Shefford, Second Prize, £10.
Francis Jordan Coleman, Hunmanby, Yorks, Certificate.
George Frederic Still, Tollington Park, Certificate.

Second Year's Students, 1890.

Arthur John Sharp, Bromley, Kent, First Prize, £25.
Charles Satchell Pantin, Blackheath, Second Prize, £10.
Frederick Gowland Hopkins, Enfield, Certificate.
William John Johnson, Shefford, Certificate.
Francis Jordan Coleman, Hunmanby, Yorks, Certificate.

First Year's Students, 1890.

Arthur Henry Leete, Wellingborough, First Prize, £50.
John Alexander Howard, Canonbury, Second Prize, £25.
Guy Eugene Manning, Hammersmith, Certificate.
Leopold Ernest Valentine Every-Clayton, Nelson, Lancashire, Certificate.

Open Scholarships in Arts.

Gerald Burton-Brown, Cambridge, 100 Guineas.
Norman Lavers, Ross, Tasmania, 50 Guineas.

Open Scholarships in Science.

Arthur George Butler, Beckenham,	bracketed equal, £61 5*s*. each.
Peter Henry Haylett, Kimberley,	
Hugh Milton Stewart, Beckenham,	

Physical Society.

Session 1891-2.

Honorary President.—S. Wilks, M.D., F.R.S.

Presidents.

Messrs. F. Colclough, B.A., M.B., A. M. Daldy, A. E. Durham, M.A., M.B., B.S., H. E. Durham, M.A., J. Fawcett, M.B., B.S., F. W. Hall, M.B., B.S., F. G. Hopkins, B.Sc., J. B. Leathes, B.A., G. Mackeson, F. Meadows Turner, B.A., A. T. Rake, A. J. Sharp, F. G. Swayne, B.A., M.B., B.S., C. F. Wakefield, H. W. Webber, M.B., T. R. Taylor, B.Sc.

Exhibits Committee.

Messrs. G. Mackeson, T. R. Taylor, A. T. Rake, A. J. Sharp.

Photographic Committee.

Messrs. E. H. Cartwright, B.A., F. Colclough, B.A., P. Hacquoil, T. S. Jackson, D. W. Samways, D.Sc., M.B., B.C.

Honorary Secretaries.—L. A. Dunn, M.S., L. E. Shaw, M.D.

PRIZEMEN FOR THE SESSION 1890-1.

The Treasurer's Prize of £8 was awarded to Mr. F. Meadows Turner, for his Essay on "Inflammation."

The Society's Prizes, First, £7, was awarded to Mr. T. R. Taylor, B.Sc., for his Paper on "Protoplasm."

The Speaking Prize of £4 for Senior Students was given to Mr. A. M. Daldy.

An Extra Speaking Prize of £3 was awarded to Mr. G. Mackeson.

CLINICAL APPOINTMENTS HELD IN THE YEAR 1891.

HOUSE PHYSICIANS.

H. Austen Smith.
F. W. Hall.
J. Fawcett.
H. Webber.
C. McG. Kitching.
J. H. Bryant.

HOUSE SURGEONS.

C. Spurrell.
A. H. Wohlmann.
R. G. P. Lansdown.
H. M. Jordan.
G. H. S. Daniell.
F. G. Swayne.
T. G. Stevens.
F. Wellford.

OBSTETRIC RESIDENTS.

T. G. Stevens.
J. H. Bryant.
J. Fawcett.
J. S. Richards.
C. R. A. Sutton.
H. Hodgson.
D. W. Samways.
W. G. Rogers.
C. G. Roberts.
A. E. Norburn.
F. S. Wood.
F. G. Swayne.

SURGEONS' DRESSERS.

W. A. Haslam.
H. L. E. Wilks.
S. J. Roberts.
G. Sichel.
A. T. Rake.
H. E. Durham.
T. B. P. Davies.
H. B. Wilkinson.
C. E. Salter.
A. W. Sheen.
C. Braithwaite.
W. A. Higgs.
J. W. Emmet.
W. D. Spurrell.
A. T. Jago.
J. E. Coulson.
S. R. H. Lucy.
R. Simons (2 months).
A. Reeves.
J. H. Muncaster.
C. M. Fleury.
F. M. Turner.
J. S. Barnes.
E. T. E. Hamilton.
T. R. Taylor.
A. M. Daldy.
P. M. May.
F. C. Young.
C. C. Elliott.
R. Gillbard.
F. G. Saffrey.
C. G. Morice.
A. H. Godson.
W. H. Fisher.
W. H. Frith.
G. T. Birdwood.
G. Mackeson.
J. D. Thomas.
R. L. Wason.
C. N. Thomas.
O. C. Worthington.
W. Bligh.
T. H. Evans.
W. T. B. Donnelly.
E. E. Frazer.
H. J. L. Wales.
T. D. Lister.
F. P. S. Cresswell.
H. St. J. Fraser.
C. E. Pollock.
E. Huntley.
F. C. Stearn.
E. J. H. Budge.
A. P. H. Lulham.
T. S. Robson.
J. O. Williams.
J. R. Evans.
A. G. Buchanan.
M. Craig.
S. G. Floyd.
W. E. Alston.
A. Allport.
E. E. B. Landon.
H. G. Biddle.

CLINICAL ASSISTANTS.

T. F. Ricketts.
A. Thomas.
H. Hodgson.
C. G. Roberts.
W. Winslow.
W. G. Rogers.
W. T. B. Donnelly.
H. Richardson.
J. W. F. Jewell.
W. E. S. Cobb.
E. G. March.
W. Carling.
C. Braithwaite.
J. E. Coulson.
E. E. B. Landon.
C. E. Pollock.
F. C. Stearn.
T. B. B. Davies.
H. B. Bolus.
F. W. Wilson.
C. E. Salter.
T. Holmes.
A. W. Sheen.
H. B. Wilkinson.

DRESSERS IN THE EYE WARDS.

J. V. Blachford.
J. A. Ward.
A. S. Wohlmann.
W. I. Robertson.
A. Thomas.
W. F. Colclough.
D. S. Long.
W. Winslow (two months).
G. Pender Smith (two months).
A. C. Elliman.
J. W. Smith.
D. W. Samways.
R. E. Williams.
E. M. Dobinson.
V. J. Hodgson.
H. B. Rygate.
J. Young.
F. A. Osborn.
H. E. Durham.
W. Carling.
J. W. F. Jewell.
H. Hodgson.
W. E. S. Cobb.
E. H. Cartwright.
W. A. Higgs.

DENTAL SURGEONS' DRESSERS.

A. A. Grosvenor.
A. C. Elliman.
E. Cornish.
J. R. R. Pollock.
A. Reeves.
A. P. Allan.
J. R. Evans.
G. H. Field.
S. C. Cresswell.
W. Winslow.
H. Hodgson.
A. L. Roper.

MEDICAL WARD CLERKS.

C. F. Wakefield.
P. Lord.
E. E. Frazer.
J. H. Muncaster.
E. P. H. Lulham.
H. J. L. Wales.
T. S. Robson.
F. C. Stearn.
C. C. Elliott.
A. H. Godson.
G. F. Birdwood.
J. R. Evans.
F. C. Young.
J. S. Barnes.
F. M. Turner.
G. Mackeson.
E. Huntley.
C. G. Gregor.
O. C. Worthington.
H. K. Rayson.
T. S. Jackson.
A. L. Allworth.
S. Croneen.
W. Bligh.
F. P. S. Cresswell.
T. D. Lister.
E. G. Evans.
C. N. Thomas.
W. H. Fisher.
W. S. Frith.
M. Craig.
A. M. Daniell.
T. H. Evans.
J. O. Williams.
A. J. Sharp.
J. D. Thomas.
W. Ashby.
H. B. Bolus.
S. E. Worts.
E. J. H. Budge.
F. W. Wilson.
A. Allport.
S. G. Floyd.
A. H. Card.
F. P. Bush.
A. G. Buchanan.
A. V. Chapman.
H. D. Eccles.
J. W. Taylor.
F. J. Coleman.
W. J. Johnson.
J. H. Godson.
W. E. Alston.
G. F. Still.
F. N. Day.
A. Shillitoe.
G. E Lockyer.
E. Hunt.
J. W. Eastment.
H. Hearnden.
E. E. S. Silver.
C. M. Greenway.
D. A. W. Martin.
W. Poole.
E. S. Dukes.
F. Hazell.
P. E. Bearblock.
C. S. Pantin.
W. C. C. Park.
H. Meyrick-Jones.
R. H. Luce.
W. J. Harris.
F. A. Robinson.
W. H. Jewell.
G. H. Field.

ASSISTANT SURGEONS' DRESSERS.

E. J. H. Budge.
F. P. Bush.
E. S. Worts.
A. Allport.
W. T. B. Donnelly.
E. T. E. Hamilton.
T. R. Taylor.
A. M. Daldy.
T. I. Mills.
H. C. Sharp.
E. E. W. Roe.
C. M. Fleury.
W. E. Alston.
S. C. Cresswell.
E. S. Dukes.
F. G. Saffrey.
A. G. Buchanan.
S. G. Floyd.
C. E. Moffat.
R. H. Luce.
E. Hunt.
G. Mackeson.
E. E. Frazer.
F. H. Edwards.
H. A. Munro.
F. C. Stearn.
L. C. Burrell.
P. E. Bearblock.
F. Hazell.
R. E. G. Phillips.
F. G. M. Philps.
G. A. Skinner.
H. A. Hinds.
G. H. Field.
P. Hacquoil.
H. J. L. Wales.
F. A. Robinson.
E. Reid.
J. R. Evans.
C. E. Cooper.
J. H. Joyce.
E. P. H. Lulham.
A. White.
T. S. Robson.
J. H. Compton.
E. Ransome.
E. Huntley.
T. S. Jackson.
W. Bligh.
M. Craig.
W. R. Grove.
C. G. Gregar.
C. W. Moorshead.
L. Slater.
J. O. Williams.
C. H. Harding.
H. Cooper.
M. Jenkins.
E. L. Adams.
J. G. B. Coleman.
A. L. Roper.
J. J. E. Biggs.
J. J. Culmer.
J. W. H. Eyre.
H. D. Eccles.
A. G. Cooley.

ASSISTANT SURGEONS' DRESSERS (*continued*).

H. G. T. Major.
W. E. de Korte.
J. W. Taylor.
F. J. Coleman.
W. J. Johnson.
G. F. Still.
A. H. Card.
A. V. Chapman.
H. P. Barker.
A. B. Creak.
D. M. Beddoe.
F. M. Day.
J. H. Godson.
W. J. Burroughs.

AURAL SURGEONS' DRESSERS.

F. N. Day.
W. H. Jewell.
H. Cooper.
C. E. Cooper.
W. A. Higgs.
R. H. Luce.
F. D. Lumley.
A. H. Meadows.
W. H. Dixon.
A. J. Sharp.
T. I. Mills.
C. H. Harding.

ASSISTANT PHYSICIANS' CLERKS.

E. E. B. Landon.
F. A. Robinson.
J. H. Muncaster.
J. R. Evans.
F. P. S. Cresswell.
W. J. Johnson.
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W. H. Peake.

DENTAL SCHOOL.

APPOINTMENTS HELD DURING THE YEAR 1891.

DENTAL HOUSE SURGEON.

F. Hopson.

ASSISTANT DENTAL HOUSE SURGEONS.

F. Haynes.
P. Harrison.
H. B. Stoner.

DEMONSTRATORS IN THE CONSERVATION ROOM.

P. Harrison.
F. W. Ta Bois.
F. J. Blewitt.
S. R. Apthorpe.
S. Keele.

CURATORS OF LABORATORY.

H. B. Stoner.
A. R. Henry.
F. P. Allworth.

DRESSERS IN THE EXTRACTION ROOM.

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R. E. Woodcock.
J. M. Tarachand.
F. H. Wallis.
W. R. Butler.
A. V. Brimmer.
F. E. Pritchard.
G. W. Whitworth.
H. P. Taylor.
E. Picnot.
F. H. Carpenter.
L. G. Musgrave.
G. Williams.
W. H. Lyne.
G. S. F. Manton.
C. H. Oram.
J. B. Parfitt.
W. R. Wood.
J. E. O. Brawn.
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E. V. Knight.

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F. Allworth.
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G. S. F. Manton.
J. M. Tarachand.
R. E. Woodcock.
F. E. Pritchard.
W. R. Butler.
A. V. Brimmer.

GUY'S HOSPITAL.

MEDICAL AND SURGICAL STAFF.

1891.

Consulting Physicians.

S. Wilks, M.D., LL.D., F.R.S.
F. W. Pavy, M.D., LL.D., F.R.S.

Consulting Obstetric Physicians.

H. Oldham, M.D.
J. Braxton Hicks, M.D., F.R.S.

Physicians.

P. H. Pye-Smith, M.D., F.R.S.
Frederick Taylor, M.D.
J. F. Goodhart, M.D.
W. Hale White, M.D.

Assistant Physicians.

G. Newton Pitt, M.D.
E. C. Perry, M.D.
L. E. Shaw, M.D.
J. W. Washbourn, M.D.

Obstetric Physician.

A. L. Galabin, M.D.

Assistant Obstetric Physician.

P. Horrocks, M.D.

Dental Surgeon.

F. Newland-Pedley, Esq.

Senior Assistant Dental Surgeon.

W. A. Maggs, Esq.

Medical Registrar.

E. W. Goodall, M.D.

Consulting Surgeons.

Edward Cock, Esq.
J. Birkett, Esq.
Thomas Bryant, M.Ch.

Consulting Ophthalmic Surgeon.

Charles Bader, Esq.

Surgeons.

Arthur E. Durham, Esq.
H. G. Howse, M.S.
N. Davies-Colley, M.C.
R. Clement Lucas, B.S.

Assistant Surgeons.

C. H. Golding-Bird, M.B.
W. H. A. Jacobson, M.Ch.
Charters J. Symonds, M.S.
W. Arbuthnot Lane, M.S.

Ophthalmic Surgeon.

C. Higgens, Esq.

Assistant Ophthalmic Surgeon.

W. A. Brailey, Esq.

Aural Surgeon.

W. Laidlaw Purves, Esq.

Instructor in Anæsthetics.

Tom Bird, Esq.

Surgical Registrar.

J. McD. Gill, M.D.

Curator of the Museum.

Dr. Shaw.

Lying-in Charity.

Dr. Galabin and Dr. Horrocks.

Dean of the Medical School and Warden of the College.

Dr. Perry.

LECTURERS AND DEMONSTRATORS.

Clinical Medicine	The Physicians and Assistant Physicians.
Clinical Surgery	The Surgeons and Assistant Surgeons.
Medicine	Dr. Pye-Smith and Dr. Taylor.
Practical Medicine	Dr. Goodall.
Surgery	Mr. Howse and Mr. Davies-Colley.
Practical Surgery	Mr. Symonds and Mr. Lane.
Operative Surgery	Mr. Symonds.
Midwifery and Diseases of Women	Dr. Galabin.
Practical Obstetrics	Dr. Horrocks.
Mental Diseases	Dr. Savage.
Ophthalmic Surgery	Mr. Higgens and Mr. Brailey.
Dental Surgery	Mr. Newland-Pedley.
Aural Surgery	Mr. Laidlaw Purves.
Diseases of the Skin	Dr. Perry.
Diseases of the Throat	Mr. Symonds.
Electro-Therapeutics	Dr. Washbourn.
Anæsthetics	Mr. Tom Bird.
Hygiene and Public Health	Mr. Turner.
Pathology	Dr. Pitt.
Morbid Anatomy	Dr. Pitt, Dr. Perry, and Dr. Shaw.
Morbid Histology	Dr. Pitt and Mr. Lane.
Bacteriology	Dr. Washbourn.
Medical Jurisprudence	Dr. Stevenson.
Anatomy	Mr. Lucas and Mr. Jacobson.
Practical Anatomy	Mr. Dunn, Mr. Targett, and Mr. Fripp.
Physiology	Mr. Golding-Bird, Dr. Washbourn, and Dr. Starling.
Histology and Practical Physiology	Dr. Starling and Mr. Tubby.
Materia Medica and Therapeutics	Dr. Hale White.
Practical Pharmacy	The Hospital Dispenser.
Chemistry	Dr. Stevenson.
Practical Chemistry	Mr. Groves.
Experimental Physics	Professor Reinold.
Biology	Mr. Beddard and Dr. Campbell.

The Hospital contains 695 beds, of which 500 are in constant occupation. Special Classes are held for Students preparing for the Examinations of the University of London, and other Higher Examinations.

APPOINTMENTS.

All Hospital Appointments are made strictly in accordance with the merits of the Candidates, and without extra payment.

ENTRANCE SCHOLARSHIPS IN SEPTEMBER, 1892.

Two Open Scholarships in Arts, one of the value of 100 guineas open to candidates under 20 years of age; and one of 50 guineas open to candidates under 25 years of age. Two Open Scholarships in Science, one of the value of 125 guineas, and another of 50 guineas, open to candidates under 25 years of age.

PRIZES

Are awarded to Students in their various years, amounting in the aggregate to more than £450.

DENTAL SCHOOL.

The Dental School is attached to the Hospital, which affords to Students all the instruction required for a Licence in Dental Surgery.

COLLEGE.

The Residential College accommodates about 50 Students in addition to the Resident Staff of the Hospital, and contains a large Dining Hall, Reading Rooms, and Gymnasium for the use of the STUDENTS' CLUB.

For Prospectus and further information apply to the Dean, Dr. PERRY, Guy's Hospital, London, S.E.

GUY'S HOSPITAL.

DENTAL SCHOOL.
1891.

Attendance of Assistant Dental Surgeons.

Mornings.

Mr. Maggs, L.R.C.P., M.R.C.S., L.D.S.Eng.
Mr. Mansbridge, L.R.C.P., M.R.C.S., L.D.S.Eng.
Mr. Harold Murray, L.D.S.Eng.
Mr. Harry Pillin, L.D.S.Eng.
Mr. G. O. Richards, M.R.C.S., L.D.S.Eng.
Mr. Rouw, L.R.C.P., M.R.C.S., L.D.S.Eng.

Afternoons.

Mr. Badcock, L.R.C.P., M.R.C.S , L.D.S.Eng.
Mr. A. E. Baker, L.R.C.P., M.R.C.S., L.D.S.Eng.
Mr. J. O. Butcher, L.D.S.Eng.
Mr. Traer Harris, M.R.C.S., L.S.A., L.D.S.Eng.

The *Dental Surgeon*, Mr. Newland-Pedley, F.R.C.S., L.D.S.Eng., attends on *Tuesdays* in the Extraction Room to see special cases.

Anæsthetists.

Dr. Cock, M.S.
Mr. Fripp, M.B., B.S.
Dr. Lancaster.
Mr. Lloyd, L.R.C.P., M.R.C.S.
Dr. J. F. Silk.

LECTURES AND DEMONSTRATIONS.

Winter Session.

Dental Surgery	Mr. Newland-Pedley.
Dental Anatomy and Physiology	Mr. Maggs.
Metallurgy	Mr. Groves, F.R.S.

Summer Session.

Dental Mechanics	Mr. Richards.
Dental Microscopy	Mr. Mansbridge.
Operative Dental Surgery	Mr. Murray.
Demonstrations of Dental Mechanics	Mr. Pillin.
Dental Tutor	Mr. Rouw.

The Dental Department of Guy's Hospital was organised as a complete School of Dental Surgery at the beginning of the Winter Session, 1889. In addition to the accommodation which already existed in the Out-patient Department for the extraction of teeth, the Governors erected special buildings—a Conservation Room, in which the operations of conservative dentistry were carried out, and a Dental Laboratory, in which Students received instruction in the construction and adjustment of artificial dentures for patients under their care. The rapid growth of the School soon necessitated increased accommodation.

The New Dental School Building is of considerable extent, and is rapidly approaching completion, and supplies all that is requisite for the practice and teaching of Dental Surgery. The new Conservation Room contains fifty Modern Operating Chairs, and it is open all day for practice; and the Special Lectures are arranged to harmonise with the General Lectures.

The New Dental Laboratory will be 65 ft. long. There will be seats for thirty students, and they will be allowed to construct and fit dentures and correction-plates for patients, thus gaining valuable experience in taking models, making "bites," and adjusting artificial teeth in the mouth. Suitable cases will be found in the Hospital, and the work done will be absolutely free of charge. Practical instruction will be given to supplement the Course of Lectures on Dental Mechanics, and Demonstrations will be held on the construction of dental splints, obturators, and vela, used in treating patients in the wards, where there are always interesting cases of surgical disorders, complicated with dental lesions.

There is an **Extraction Room** for ordinary cases, and a **Gas Room** for operations under Anæsthetics, which are administered daily to such patients as the dental officers recommend. Cases suitable for conservative treatment are transferred to the Conservation Room at the discretion of the Assistant Dental Surgeons.

The Teaching Power of the School has also been augmented by the appointment of four additional Assistant Dental Surgeons, who attend in the afternoons.

Every Facility is afforded to students desiring to take medical qualifications in addition to the L.D.S., and in their case the dental curriculum can be spread over the whole general course.

Continuous Instruction is afforded by means of a separate morning and afternoon staff of Dental Officers. The lectures are given at hours convenient to students. Ten clinical demonstrations are given weekly by members of the Dental Staff.

A Clinical Demonstration is given at 11 a.m. by the Assistant Dental Surgeon for the day on one of the various methods of filling, the preparation of roots for pivots, and the adaptation of porcelain and metal crowns to broken-down teeth. Electric Motors and Mallets are employed in these demonstrations, and a Dynamo has been installed.

Prizes have lately been established, and will be awarded for the first time in the present academical year. For First Year's Students, a Prize of £10; for Second Year's Students, Prizes of £15 and £10—the latter being given for Operative Dental Surgery.

The Fee for the General and Special Lectures and Hospital Practice required for the L.D.S.Eng. is £90 on entrance, or 90 guineas by instalments. The Fee for L.R.C.P., M.R.C.S., L.D.S.Eng. is £180 on entrance, or 180 guineas by instalments. Further information with Prospectus can be obtained from the Dean, Guy's Hospital, S.E.

ASTLEY COOPER PRIZE.

The next Triennial Prize of Three Hundred Pounds,

Under the Will of the late SIR ASTLEY P. COOPER, Bart.,

WILL BE AWARDED TO

THE AUTHOR OF THE BEST ESSAY OR TREATISE ON

"*The Anatomical Distribution of the Lymphatic Vessels and the Physical Forces concerned in the movement of the Lymph.*"

THE Conditions annexed by the Testator are, "That the Essays or "Treatises to be written for such Prize shall contain original experi-"ments and observations, which shall not have been previously pub-"lished, *and that each Essay or Treatise shall* (as far as the subject "shall admit of) *be illustrated by preparations and by drawings*, which "preparations and drawings shall be added to the Museum of Guy's "Hospital, and shall, together with the Work itself and the sole and "exclusive interest therein and the copyright thereof, become hence-"forth the property of that Institution, and shall be relinquished and "transferred as such by the successful candidate."

And it is expressly declared in the Will "That no Physician or "Surgeon, or other officer for the time being, of Guy's Hospital or of "St. Thomas's Hospital, in the Borough of Southwark, nor any person "related by blood or affinity to any such Physician or Surgeon, for "the time being, or to any other Officer for the time being in either "of the said Hospitals, shall at any time receive or be entitled to "claim the Prize." But, with the exceptions here referred to, this Prize is open for competition to the whole world.*

Candidates are informed that their Essays, either written in the English Language, or, if in a Foreign Language, accompanied by an English translation, must be sent to Guy's Hospital on or before January 1st, 1895, addressed to the Physicians and Surgeons of Guy's Hospital.

Each Essay or Treatise must be distinguished by a Motto, and be accompanied by a sealed envelope containing the name and address of the Writer. None of the envelopes will be opened except that which accompanies the successful Treatise. The unsuccessful Essays or Treatises, with the illustrative preparations or drawings, will remain at the Museum of Guy's Hospital until claimed by the respective writers or their agents.

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* The Prize cannot be awarded to any Essay which is the joint production of two or more authors.

GUY'S HOSPITAL REPORTS FOR 1890.

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3. Sarcoma of the Tongue. By J. H. Targett, M.S.
4. An Analysis of the Cases of Chorea which have occurred in the Hospital during Eleven Consecutive Years. By E. W. Goodall, M.D.
5. A Case of Fracture of the Coracoid Process uncomplicated with other Injury, with a Study of the Lesion. By R. Clement Lucas, B.S.
6. On Primary Malignant Disease of the Liver. By W. Hale White, M.D.
7. The Pathology of Acute Infective Periostitis and Acute Necrosis of Growing Bone. Being the Report of an Investigation carried on in the Workrooms of the Royal College of Surgeons of England and the Pathological Institute at Leipzig. By A. H. Tubby, M.S.
8. Chiefly concerning Bruits. A Lecture on Pathology from a Course upon "Points in the Pathology of Diseases of the Heart." By James F. Goodhart, M.D.
9. A Case of Glanders, with the Results of Cultivation and Inoculation Experiments. By J. W. Washbourn, M.D.
10. On the Pathology and Prognosis of Pernicious Anæmia. By W. Hale White, M.D.
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12. Congenital Wry-Neck (Caput obstipum congenitale; Torticollis congenitalis); with Remarks on Facial Hemiatrophy. By C. H. Golding-Bird.
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16. Abnormalities observed in the Dissecting Room of Guy's Hospital during the Sessions 1888—1890. By L. H. Dunn, M.S., J. W. Washbourn, M.D., and J. H. Targett, M.S.
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18. List of Specimens added to the Pathological Museum during the years 1888–89. By G. Newton Pitt, M.D., and Lauriston E. Shaw, M.D.

List of Pupils who have passed the Examinations of the several Universities, Colleges, &c., 1889.
List of Prizemen and Medallists, 1888–89.
List of Pupils who have received Appointments at Guy's Hospital, 1889.

J. & A. Churchill, New Burlington Street.

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